Industrial Medicine

for

Physicians and Surgeons in Industry

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Analysis of 1000 Deaths
—Among Railroad Employees Subject to Periodic Examination—

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—Selection and Maintenance—

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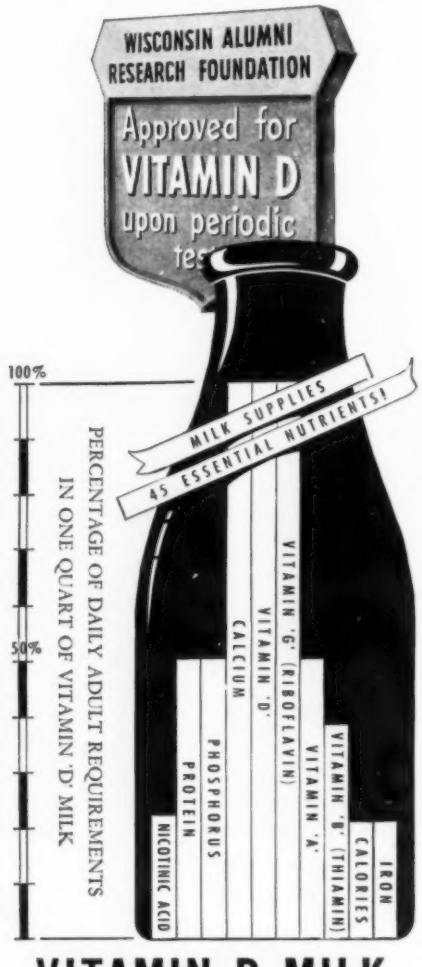

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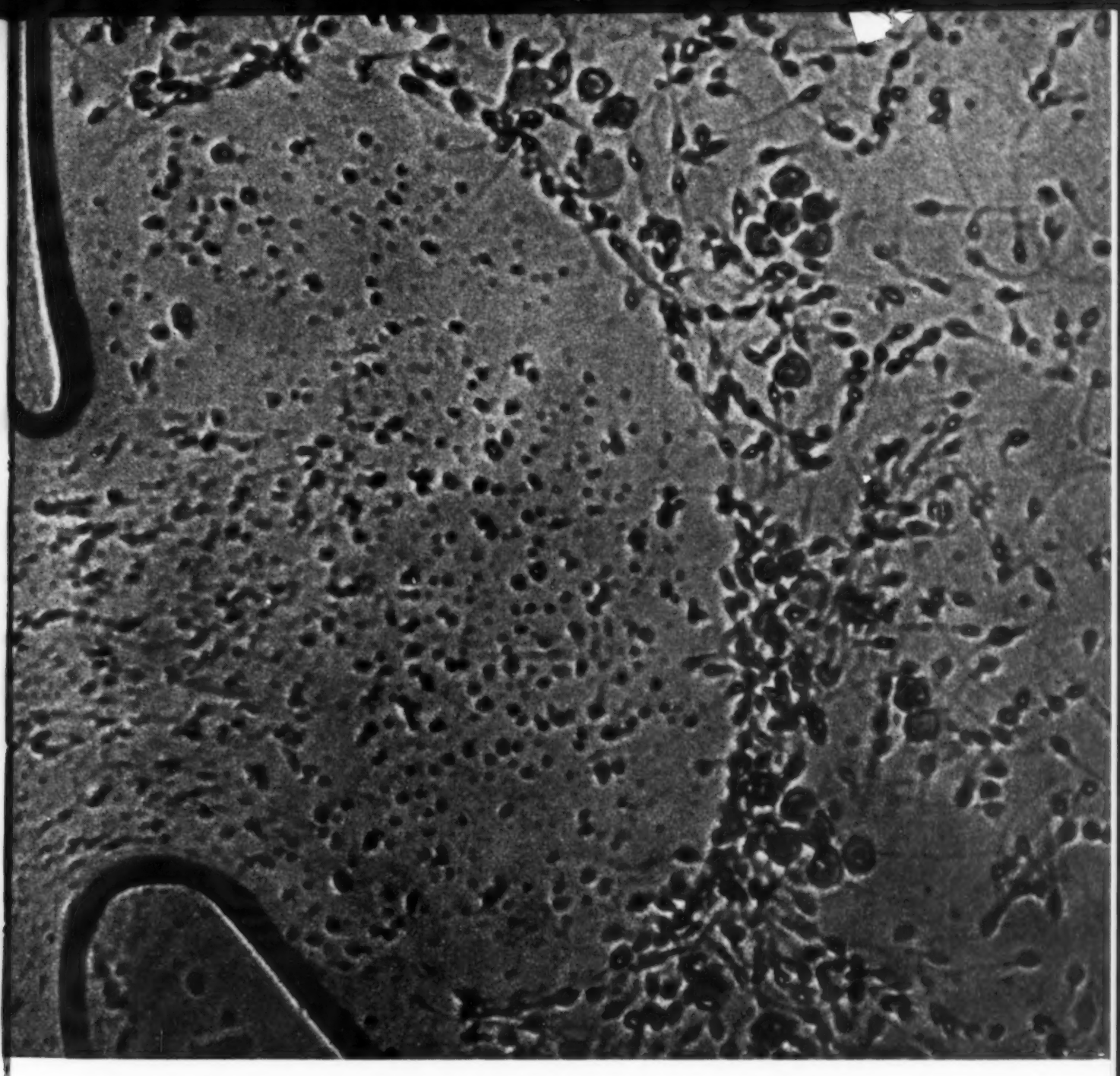

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THE mode of action of sulfanilamide, as DRS. KEY and LEMBECK remark (page 493), may not be known, but the range of action is being constantly extended — as these authors exemplify in their "Local Use, in Compound Fractures and Other Wounds." This is the first of four papers from the recent Fifty-First Annual Meeting of the AMERICAN ASSOCIATION OF RAILWAY SURGEONS. The others are DR. HARDT'S "Gastroscopy as an Aid in Differential Diagnosis" (page 497); DR. SEGALL'S "The Ageing Process; Statistical Notes on Coronary Stenosis and Thrombosis" (page 521); and DR. SHINGLE'S "Tick Fevers" (page 526).

COLONEL TUTTLE'S "Personnel in Industry" (page 500) emphasizes the importance of visual efficiency. DR. CLARK, on page 503, talks of "Serious Conditions of the Eyes and Ears Following Minor Ailments." DR. BELKNAP'S "Criteria for the Diagnosis of Lead Poisoning" was read at the A. A. I. P. & S. convention in New York, last June. DR. MYERS' "Analysis of 1000 Deaths" (page 509) is from the Seventeenth Annual Meeting of the ASSOCIATION OF SURGEONS OF THE NEW YORK CENTRAL SYSTEM, as is also DR. HERRICK'S "Diagnostic Factors in Circulatory Disease" (page 513). DR. WOODY'S "Industrial Medicine as a Specialty" (page 510), and DR. BOLAND'S "History of Fractures" (page 517), are from the Third Annual Meeting of the GEORGIA ASSOCIATION OF INDUSTRIAL SURGEONS at Atlanta, last month.

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INDUSTRIAL MEDICINE, The Journal of Occupational Diseases and Traumatic Surgery, entered as second class mail matter at the Post Office at Beloit, Wisconsin, under the Act of March 2, 1879. Published on the fifth of each month by INDUSTRIAL MEDICINE PUBLISHING COMPANY, Beloit, Wisconsin. R. F. COLLINS, President; A. D. CLOUD, Managing Editor; A. G. PARK, Vice-President; CLINTON F. KARSTAEDT, Secretary-Treasurer and Business Manager. PUBLICATION and EXECUTIVE Offices: 413 Pleasant Street, Beloit, Wisconsin; EDITORIAL Office, 540 No. Michigan Ave., Chicago, Illinois. Subscription $5.00 per year in the United States; $5.50 per year in Canada; $6.00 per year in other countries. Single copies 50 cents. Title Registered in U. S. Pat. Off.

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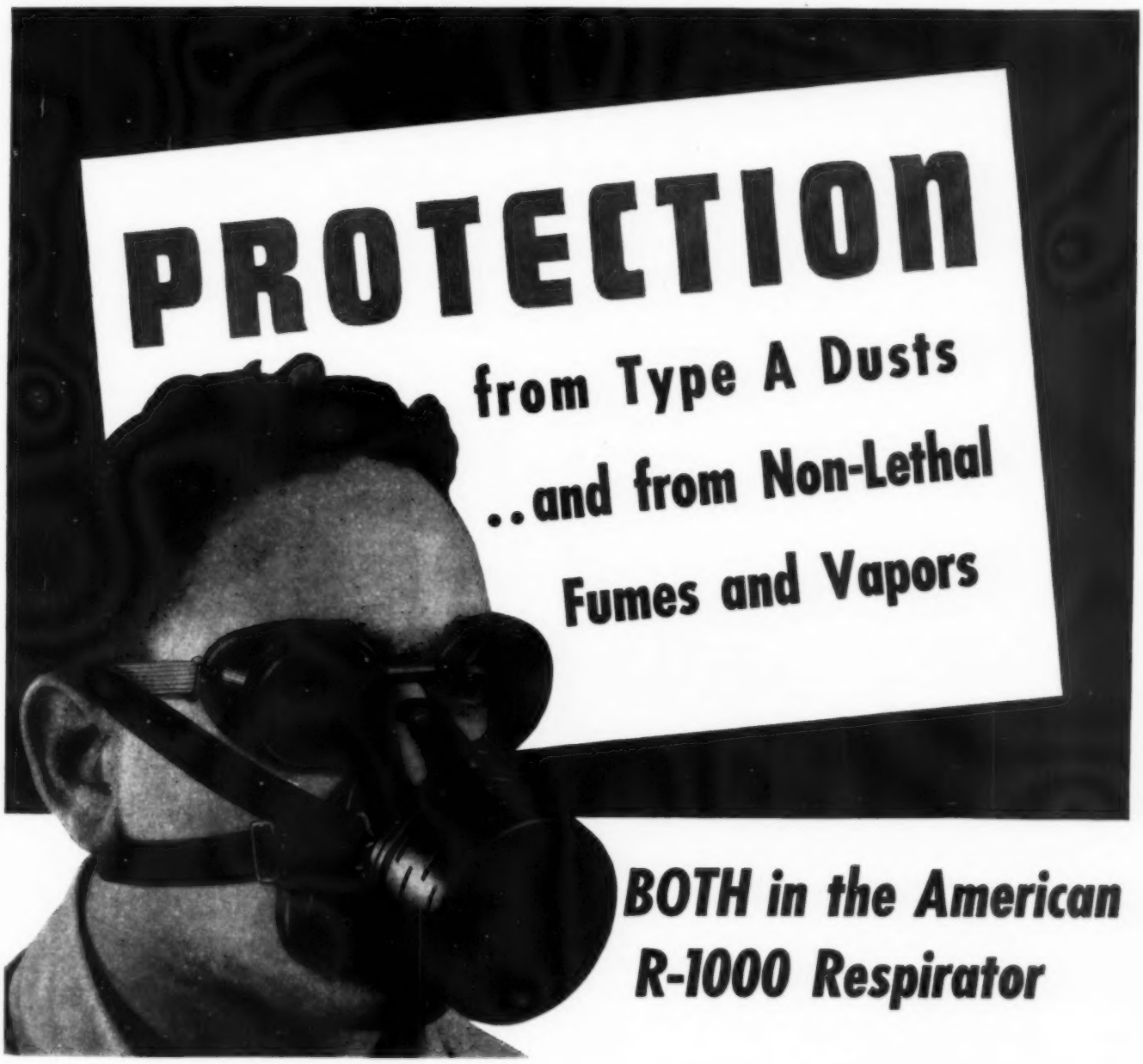

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Published under supervision of the Ways and Means Committee of the New York State Society of Industrial Medicine. Editorial office, F. E. Redmond, Managing Editor, 361 Delaware Avenue, Buffalo, N. Y. Articles appearing herein do not necessarily reflect the opinion or policy of this journal nor of the New York State Society of Industrial Medicine.

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—*Continued from page 3.*

DRS. PAUL and JOHN FORGRAVE, in their "Comomn Cold—Prophylaxis by the Oral Route" (page 530) contribute another to the growing number of articles on this ever timely subject. And then we quote GLADYS JAHNCKE, R.N., on "Home Hygiene Classes in Industry" (page 533), and DR. PAUL A. BREHM on "Physical Examinations of Industrial Workers" (page 535).

APROPOS of the place and importance of the skilled worker in national defense, AIR HYGIENE FOUNDATION, in *Foundation Facts* for September, 1940, quotes the "Armorer's Song," from "Robin Hood," as follows: "The sword is a weapon to conquer fields, I honor the man who shakes it. But naught is the man who the broadsword wields, compared to the man who makes it!"

"INDUSTRIAL Workers of 1960," by DR. CAREY P. MCCORD, in INDUSTRIAL MEDICINE for April, 1940, contained some forecasts of the results of the 1940 Census which, although they were based on current trends, found many readers loath to believe in their possibilities. It is interesting to note that the published previews of the Census results substantiate DR. MCCORD'S prophecies with astonishing fidelity. One of the best of the previews was that one in *United States News* for August 23, 1940.

The Symposium on the Practical Value and Application of Industrial Health Work, to be held by the recently organized AMERICAN CONFERENCE ON INDUSTRIAL HEALTH on November 14, as announced on page 525, is deserving of special attention, and a special effort to be present at the sessions. The Program promises it will be worth while.

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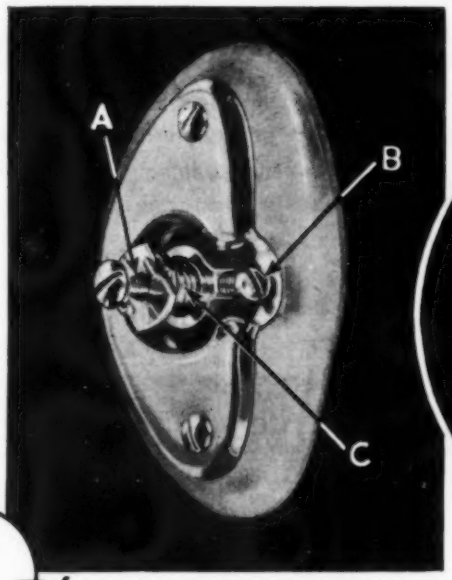

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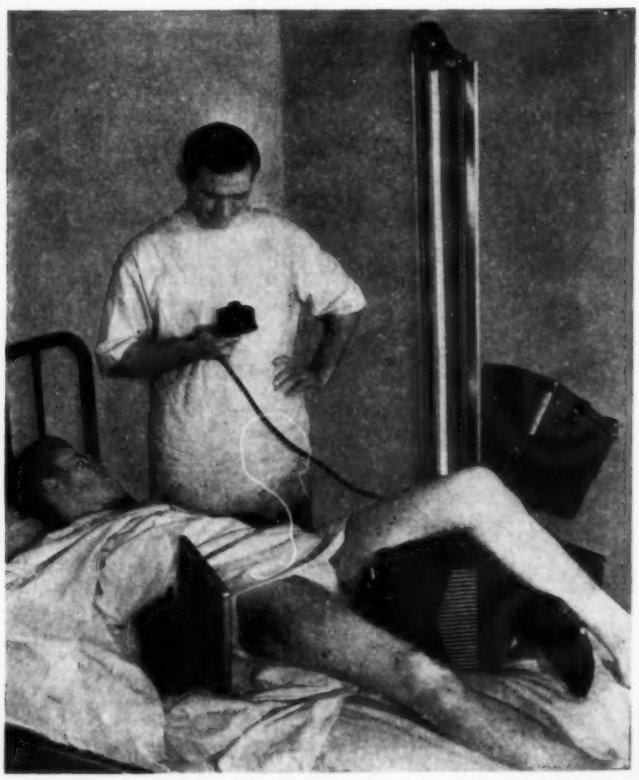

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The Journal of Occupational Diseases and Traumatic Surgery
With which are consolidated "The Industrial Doctor" and "International Journal of Medicine and Surgery."

The Science, the Law and the Economics of Industrial Health

Volume 9 OCTOBER, 1940 Number 10

Local Use of Sulfanilamide

—*In Compound Fractures and Other Wounds*—

J. Albert Key, M.D. and Joseph A. Lembeck, M.D
St. Louis, Missouri

SULFANILAMIDE may be obtained in tablet form for oral administration or as a white, crystalline powder. This crystalline powder is soluble in water to about 1%—that is, about 1000 milligrams per cent. When the drug is administered orally in therapeutic doses it has been found that the drug enters the blood stream and is bacteriostatic for certain organisms, especially the beta-hemolytic streptococcus.

In order to combat infections which are susceptible to sulfanilamide it is necessary to maintain a concentration in the blood of approximately 10 milligrams per cent. This requires frequent administration of the drug over the 24-hour period and the administration must be continued as long as the infection is active. Under such conditions the drug inhibits the growth of only certain organisms which are susceptible to it and thus is highly selective. Furthermore, it has been shown that the drug does not confer any immunity on the patient to whom it is given, but that it acts directly upon the bacteria and that in order to affect bacteria it must come in contact with them in the animal body or in culture medium.

The mode of action of sulfanilamide is not known. In the concentrations which are obtained in the blood by therapeutic doses the drug is not bactericidal, but it merely inhibits the growth of the organisms and apparently converts a pathogenic streptococcus into a non-pathogenic form. The bacteria may then be destroyed by the defense mechanism of the body. It is thus evident that when given orally the drug is highly selective in its action and its principal value is in combating certain general infections which are susceptible to it and it is of little value in treating infections due to staphylococci.

Since sulfanilamide is soluble in water and in the body fluids to approximately 1% by weight, a relatively high local concentration of the drug can be obtained if the drug in powder form is placed directly in a wound and the wound is closed, because the dry powder will be dissolved in the fluids which collect in the wound. Under such conditions the local concentration of the drug is from 80 to 100 times that which can be maintained in the blood stream by therapeutic doses. The drug not only saturates the fluid in the wound, but it diffuses out into the surrounding tissues and is slowly absorbed and excreted.

While dilute solutions of sulfanilamide affect only a few susceptible bacteria, stronger solutions not only are more active against susceptible bacteria, but also affect others which are relatively immune to the dilute solutions. Consequently, when powdered sulfanilamide is placed directly in a wound we have a drug which not only is bacteriostatic for susceptible bacteria, but is also actually bactericidal for susceptible bacteria and is bacteriostatic for a wide variety of organisms. Among the latter are the pathogenic staphylococci and the gas bacillus of Welch. It is thus readily understandable that, while therapeutic doses of the drug by mouth might have relatively little effect in preventing a local infection, the same drug implanted in a wound might have a very definite effect.

It is further to be noted that the action of the drug on bacteria varies inversely with the number of bacteria present and is inhibited by peptones, which are products of the breaking down of protein. Consequently, in an area which contains large numbers of bacteria and in which there is considerable tissue destruction we have a condition favorable to bacterial growth and unfavorable to the action of sulfanilamide. It is also to be noted that sulfanilamide in high concentrations has a

From the Department of Surgery of the Washington University School of Medicine and the Missouri Pacific Hospital Association. Presented at the Fifty-First Annual Meeting of the American Association of Railway Surgeons, Chicago, September 16, 1940.

definite neutralizing power on the toxins of both staphylococci and B Welchii. For this reason destruction of tissues by the toxins of these bacteria is inhibited in the presence of high concentrations of sulfanilamide. Therefore, it seems logical to thoroughly debride contaminated wounds and then implant dry sulfanilamide powder in the wounds in order to prevent infection.

This was first done by Jensen, Johnsrud and Nelson,[1] who used the dry powder in compound fractures and reported that by its use and with no other alteration in technique they were able to reduce the incidence of infection of compound fractures from 28% to less than 5%. The beneficial effect of the local implantation of the drug in compound fractures was confirmed by Key.[2]

Naturally, the implantation of considerable amounts of a drug of this type in the tissues aroused some criticism, and Bricker and Graham[3] noted that wound healing appeared to be delayed in dogs which were given therapeutic amounts of sulfanilamide. On the other hand, Key and Burford[4] showed that when sulfanilamide was implanted in experimental fractures in rabbits and these fractures were compared with similar control fractures on the other leg of the same animal in which no sulfanilamide had been used there was no demonstrable difference in the rate of healing of the bone on the two sides, nor was there any demonstrable difference in the rate of healing of the soft parts.

Further investigations demonstrated that the local implantation of sulfanilamide powder was well tolerated by joints, tendon sheaths, subcutaneous tissues, muscles and body cavities, and that it did not appreciably delay the healing of wounds in experimental animals (Key, Frankel and Burford).[5] In the same paper it was noted that sulfanilamide could be used with impunity in open infected wounds and that it did not seriously interfere with the healing of such wounds. It was also noted that sulfanilamide powder could be used with freedom in clean operative wounds, but that the powder should be sterilized before being placed in such wounds, because, since it did not actually kill either staphylococci or B Welchii, there was a possibility that unless it was sterilized, pathogenic bacteria might be placed in the wound with the unsterile powder.

When other sulfanilamide compounds appeared, it became advisable to study the local action of these compounds and to determine, if possible, whether or not any of the newer compounds was more effective locally in preventing infection than was the parent drug, sulfanilamide.

Key and Frankel[6] experimented with sulfanilamide, sulfapyridine and sulfamethylthiazol and found that these drugs in powdered form were equally well tolerated by the tissues and that they did not materially affect the rate of healing of wounds in experimental animals. They were not able to compare the effects of these drugs on experimental infections, because they were not able to produce uniform infected wounds in experimental animals. As a result of their observations it was concluded that sulfanilamide was the preferable drug because of its greater solubility, it being approximately four times as soluble in the tissue fluids as was sulfapyridine or sulfamethylthiazol. Consequently, this drug is used because it is possible to obtain a more highly concentrated solution in the wound.

It is to be emphasized that sulfanilamide is not a cure of infection in compound fractures, but that its principal value lies in the fact that when it is placed in the wound after the wound has been debrided and before it is closed, it will considerably lessen the chances that this wound will become infected, even though a few pathogenic bacteria may be left in the wound. Under such conditions the sulfanilamide appears to inhibit the growth of the bacteria and they are then destroyed by the normal defense mechanisms of the body. It is thus evident that not only should the wound be debrided, but also that all foreign material should be carefully removed from the wound. Since the sulfanilamide is gradually absorbed and disappears in from two to three days, if foreign material containing bacteria is left in the wound, these bacteria may later begin to grow and may cause infection after the wound has healed and after the sulfanilamide has disappeared.

AT THE present time it is our belief that compound fractures should be treated as follows:

If severe hemorrhage is present this should be stopped either by a tourniquet or pressure dressing. The limb should be straightened if it is markedly deformed and if the fracture involves a lower limb this should be splinted before the patient is transported. The wound should be covered with as sterile a dressing as is procurable at the time. but as a rule no attempt should be made to reduce the fracture or to pull the skin back over the fragments of bone, which may be protruding from the tissue. There are, of course, exceptions to this rule.

It is important that the patient should be moved to a hospital where he will receive expert treatment as soon as possible after the injury, because time is just as important in the treatment of compound fractures with sulfanilamide as it is without it. During the interval between the time when the patient is put in an ambulance and transported to the hospital he should be made as comfortable as possible and moved as little as possible. If the lower extremity is involved, transportation should be by ambulance if possible. If it is necessary that the patient be transported over a considerable distance which will involve a period of several hours. it is advisable if sulfanilamide powder is available to sprinkle the wound thoroughly with this powder before the dressing is applied.

When the patient arrives at the hospital the first consideration is still the general condition of the patient. If he is in good general condition he should be operated upon as soon as possible after admission. If shock is present this should be treated immediately. The patient should be put in a warm bed in shock position, blood loss should be replaced by transfusion or by glucose and acacia, and sufficient opiates should be given to

relieve the pain. It is also helpful to inject 1% novocain into the tissues around the wounds in order to lessen the pain and this also tends to lessen the shock. X-ray examination should be deferred until the shock treatment is instituted and well under way. Then, if necessary, portable x-rays may be made if a portable machine is available and if the patient's general condition makes it unwise to move him to the x-ray room.

As soon as the patient's general condition has improved sufficiently he should be taken to the operating room and prepared for the definitive treatment of the compound fracture. Either local or general anesthesia may be used. After the anesthetic has been administered the injured limb should be thoroughly cleaned and draped. We scrub the skin around the wound with green soap and water and then wash the wound with green soap and water, flushing it several times, and then wash the skin with alcohol and ether and apply some skin antiseptic—usually tincture of merthiolate or tincture of mercresin. The limb is then draped in such a manner that the fractured area is freely exposed and in a manner that will permit adequate manipulation of the limb for reduction of the fracture after the site of the fracture has been exposed.

The technique of the operation is as follows: The margins of the wound should be excised, removing as little skin as possible, but removing a narrow strip entirely around the wound. The wound should then be enlarged upward and downward in order to expose the site of the fracture. As a rule, the operation is not done under a tourniquet, because the manipulation necessary for the application of a tourniquet may further increase the shock and may add to the damage of the soft tissues. As the wound is enlarged all blood vessels which are cut or which are found torn in the wound are promptly clamped and ligated with fine silk. It is important that the patient lose as little blood as possible. All grossly devitalized tissue is then excised and after the fracture has been exposed the wound is thoroughly irrigated with salt solution, using a considerable amount of fluid and forcing it into all the recesses of the wound. As a rule, we use an irrigating can with a soft rubber tube and by squeezing the tube we can increase the pressure of the stream sufficiently to thoroughly cleanse the wound. This not only serves to wash out and to expose foreign material which may be embedded in the wound, but also to float up devitalized tissue which can be excised as soon as it is identified. We do not object to the opening of fascial spaces in this manner, as it is more important that they be opened and cleaned than that they not be inspected and possibly harbor foreign material.

If the ends of the fragments are grossly contaminated by having been forced into the dirt or by having come in contact with other material, such as grease, they should be excised down to a point where clean bone is exposed. This excision of the ends of the fragments is most conveniently done with heavy bone-cutting forceps, but if these are not available osteotomes or a saw may be used. In a compound epiphyseal separation where the end of the shaft has been forced into the dirt we have not excised the end of the shaft, but have thoroughly scrubbed this with a hand brush and green soap and water before reducing the epiphyseal separation.

After the wound has been thoroughly washed it is gone over again carefully and any remaining devitalized tissue and any foreign material which can be identified in the wound are removed. Likewise, any other bleeding points are secured and ligated. The wound is then rendered as dry as possible and is reviewed for anatomical structures which may have been severed by the injury and which may need repair. These include muscles and tendons and nerves and it is our practice to suture these at the time of the debridement rather than to close the wound and expect to go in later and perform the necessary suture. For the muscles and tendons we use medium silk and for the nerves we use fine silk. We prefer silk to catgut, because we believe that the catgut causes more of an initial reaction and that if a compound fracture is going to be infected it is usually infected within the first few days after the injury. We do not believe that the non-absorbable sutures are particularly harmful and we believe that there is less danger of their causing an early reaction which may lead to an infection.

The displacement of the fragments is now corrected—usually by traction on the extremity and by manipulation of the ends of the fragments with bone holding forceps or a bone skid. If the contour of the ends of the bones is such that the position of the fragments is relatively stable after the displacement is reduced, no internal fixation is used. On the other hand, if the fracture is oblique or comminuted we no longer hesitate to use internal fixation where it is indicated, using either stainless steel wire or stainless steel plates and screws or long stainless steel screws in certain cases of oblique fractures. We believe that the added immobilization afforded by the internal fixation more than compensates for the extra risk incurred by placing of foreign material in the wound, especially since we have available stainless steel and vitallium, which seem to be equally well tolerated in the tissues and cause little initial or delayed reaction.

In comminuted fractures the problem as to how to deal with the loose fragments must be met. In the past, before we used sulfanilamide, our practice was to remove all fragments which did not appear to have sufficient periosteal attachment to maintain their blood supply and keep them viable. Since we have been using sulfanilamide we leave more fragments in and frequently replace completely detached fragments, because they aid in maintaining the contour of the bone and we believe that, although they may die, they will be incorporated in the callus and will be revivified by creeping replacement and thus serve as autogenous bone grafts.

After the fracture has been finally reduced the wound is gone over again for smaller blood vessels which may still be bleeding. If these are

found they are ligated. The wound is then sprinkled liberally with sulfanilamide powder, care being taken to see that some of the drug is placed in the deeper recesses of the wound. In the treatment of compound fractures we use a considerable amount of the powder, frequently using 10 or 15 grams or more. Apparently this relatively large amount of the drug has not caused any ill effect to the patients in whom we have used it. As a rule, we attempt to get some of the powder into the marrow canal before the fracture is finally reduced and immobilized by internal fixation, if such is to be used.

The wound is then closed with a single layer of silkworm gut sutures. These are deep sutures including the skin and subcutaneous tissues, no effort is made to close the fascia, nor is any effort made to close the wound in layers. We believe that it is important that the skin be closed over the fracture if possible, as, if the fracture site is left exposed, even though infection may not supervene, healing will be considerably delayed. Consequently, in instances where there has been considerable loss of tissue we make lateral incisions on either side of the wound and pull the skin and superficial tissues over as a sliding graft, either covering the defect on the side of the limb with free split grafts or covering the defect with vaseline gauze and letting it heal by granulation.

After the wound is closed the skin is painted with tincture of merthiolate or tincture of mercresin in order to lessen the tendency to the formation of stitch abscesses. A single layer of vaseline gauze is then placed over the wound. This prevents the dressing from adhering to and sealing the wound and permits the escape of fluid from the wound during the first few days after the operation. This is covered with a dry gauze dressing and the limb is immobilized in a plaster-of-Paris cast which is applied over a moderate amount of padding. The patient is then put to bed and general treatment is given as indicated. Unless there is some necessity for inspecting the wound, the wound is not dressed until the cast is changed. As a rule, the silkworm gut sutures are well tolerated and do not have the same tendency to cause stitch abscesses as does silk or catgut.

We have used this method in wounds which are not more than 24 hours old, preferably in wounds which are not more than 12 hours old. The time element may be extended to 24 hours in wounds which are not complicated by severe damage to the soft tissues. In certain instances where the limb is relatively clean and where the fracture appears to have been compounded from within—that is, by the bone being forced out through the skin—we have performed the debridement, but have omitted the thorough washing out of the wound and have depended upon the sulfanilamide to prevent infection. If the fracture involves the joint, the joint is opened freely, thoroughly washed out with salt solution and then sulfanilamide powder is implanted directly into the joint cavity.

In instances where the patient does not arrive at the hospital until after infection has set in, we have opened the wound widely and removed obviously devitalized tissue and sprinkled the surface liberally with sulfanilamide and then packed the wound open with vaseline gauze. In instances where frank infection of the bone is present we have continued to treat the wound by the Orr method, but have removed the vaseline gauze through a window in the cast every few days and have sprinkled the wound with sulfanilamide and redressed the wound. In this manner we believe that we have considerably lessened the amount of infection present and have been able to hasten the healing of some of our infected bone cases. In other instances where we have operated for old osteomyelitis we have continued to use the Orr method, but have added a liberal amount of sulfanilamide to the original dressing and have then not dressed the wound until approximately two weeks afterwards, depending upon the amount of discharge from the wound. We believe that the use of sulfanilamide powder in these cases has lessened the amount of infection and has decreased the amount of drainage and has aided in the healing of the wound.

Not only do we use sulfanilamide routinely in the treatment of compound fractures, but we use it even more frequently in the treatment of lacerated wounds and in puncture wounds from which foreign bodies have been removed and the wounds then sutured. Likewise, we use it in compound dislocations and lacerated wounds involving tendons.

One of us (J.A.K.) now uses sulfanilamide powder almost routinely in clean operative surgery and in approximately 100 almost consecutive operations in which a small amount of sulfanilamide powder was sprinkled in the wound before it was closed the results have been satisfactory. In using the powder in clean operative wounds less of the drug is used than is indicated in compound fractures—usually from two to four grams of the powder, the amount depending upon the size of the wound and the amount of tissue damage inflicted during the operation.

Likewise, since the introduction of sulfanilamide we have grown considerably more bold in our treatment of non-union and infected compound fractures and have operated such cases much sooner than we would have done otherwise. In this group we have had one infection. That was a severely infected compound fracture of the ankle which had united in a position of marked equinus and varus. This deformity was corrected by a wedge osteotomy three months after drainage had ceased. This was performed through an incision on the outer side of the limb after the removal of considerable scar tissue. The infection had been severe on both the inner and outer sides of the ankle. This patient was immobilized in a plaster-of-Paris cast after the operation with the foot in a satisfactory position. The cast was not removed until 14 days after the operation. When the cast was removed it was found that a large hematoma had collected in the wound, that the skin edges had separated and that the hematoma was infected. There had been no elevation of the patient's temperature, but the wound con-

tinued to drain for several weeks until it was reopened and a silk suture was found and removed. With this exception our results have been satisfactory.

The case cited in the preceding paragraph is evidence that the protective action of sulfanilamide in the wound is relatively short-lived. In other words, it is probable that in this case the sulfanilamide had disappeared from the tissues before the infection began in the hematoma.

In using sulfanilamide in compound fractures we have not been greatly concerned as to whether or not the powder was sterilized before use, because we believe that one is fairly safe in assuming that no pathogenic bacteria will be carried in with the powder. However, if one is to use the drug in clean operative wounds one should be relatively certain that the powder is sterile before it is placed in the wound. Fortunately, it is now possible to secure sulfanilamide powder which is put up in sterile form in ampoules. Under these conditions it can be used with impunity.

Lacking ampoules of sterile sulfanilamide powder, we have routinely sterilized the powder by placing it in a small flask, covering the mouth of the flask with four layers of gauze and two layers of paper, and then placing the flask in the autoclave under 15 pounds pressure for 30 minutes. It is then placed under the vacuum for about 10 minutes and then dried. When sulfanilamide is sterilized in this manner it will be found that the powder usually forms aggregates or lumps and that at times it becomes slightly yellowish in color.

It is stated that one should not use the powder if it is yellowish in color. We have used it repeatedly and have noted no ill effects. It is probable that when the powder is discolored some of the sulfanilamide is destroyed. However, apparently whatever substances are formed are not detrimental to the healing of the wound and sufficient unchanged sulfanilamide appears to be left in the powder so that it is still useful. It is preferable, however, that the powder be white and finely divided when it is used. We have found that by sterilization in the manner described above reltively little of the sulfanilamide is destroyed and if a ten-milligram per cent solution of this autoclaved powder is compared with a ten-milligram per cent solution of the dry powder before sterilization, a determination of the amount of sulfanilamide in the two solutions shows relatively little difference.

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Gastroscopy

—As an Aid in Differential Diagnosis—

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THE introduction and enthusiastic elucidation by Dr. Schindler of the Wolf-Schindler gastroscope has helped to popularize endoscopic examination of the stomach. The simplicity and harmlessness of the procedure have given added confidence in gastroscopy as an aid in diagnosis.

The proper preparation, both medically and mentally, of the patient practically rules out all discomfort. Following the examination, most of the patients volunteer the information that the procedure is not uncomfortable. My experience is in accord with Schindler, Moersch, and others, in that in more than 90% of the cases a satisfactory examination of the interior of the stomach can be obtained.

In making the gastroscopic examination we must bear in mind that there are two small portions of the stomach which cannot be visualized with the gastroscope. One portion is along the lesser curvature of the antrum, hidden from view by the angulus, and the other is a narrow strip along the posterior wall where the window of the instrument is in contact with the wall of the stomach. Failures may occur in a small percentage of cases where spinal deformities exist, in extremely short-necked individuals who cannot successfully flex the head backwards, and also, but rarely, in cases of extreme nervous tension. Contra-indications to gastroscopy may be summed up very briefly as follows: esophageal disease, carcinoma involving the cardia with obstruction, extreme hypertension, aneurism, and cardiospasm. I do not agree with Moersch in his statement that active pulmonary tuberculosis is a contra-indication, since we have to date gastroscoped with no untoward effects over 100 patients who have active pulmonary tuberculosis.

I will now discuss the use of the gastroscope in cases of gastritis, ulcers, benign tumors, carcinomas, and gastric hemorrhage, punctuating the discussion with case histories.

Gastritis

GASTROSCOPY has been an invaluable aid in the diagnosis of inflammatory lesions of the stomach which apparently have not produced enough deformity for Roentgen diagnosis. The recognition of gastritis has aided materially in explaining many epigastric symptoms which heretofore had been placed in the category of gastric neuroses. Although the first description of an acute inflammation of the gastric mucosa was published in 1838 by Beaumont, no emphasis was

Presented at the Fifty-First Annual meeting of the AMERICAN ASSOCIATION OF RAILWAY SURGEONS, Chicago, September 17, 1940.

placed on this as a disease entity until recent years. For practical purposes, we might divide gastritis into three general types, designated as acute, superficial or catarrhal; hypertrophic; and atrophic.

In the acute gastritis, the rugae are swollen, reddened at times to a beefy red, and appear edematous. This is usually accompanied by an exudate, often pus-like in appearance, which occurs on and between the folds. The inflamed gastric mucosa has a tendency to bleed easily, and for this reason is often referred to as an acute hemorrhagic gastritis. Small erosions may occur. Epigastric pain and tenderness associated with belching are typical symptoms of this type of gastritis, at times similar to an ulcer-like syndrome.

Case 1. A typical case history of a patient with this form of gastritis is as follows. The patient was admitted to the hospital March 13, 1940, suffering from epigastric pain, general in character, belching, and distress for over one year. The gastric analysis showed 13 degrees free HC1 and 37 degrees total acidity. A barium meal showed a constant narrowing and elongation of the pyloric portion of the stomach which was diagnosed as either carcinoma or antral gastritis. Gastroscopy report on March 15, 1940, was as follows: the mucosa throughout the stomach was swollen, purplish red in color, with an almost complete obliteration of rugae. On the mucosal surface were patches of purulent mucus, greenish in color. The diagnosis was acute gastritis.

In the hypertrophic gastritis, the mucosal folds are also swollen, the mucous membrane is velvety in appearance, the highlights are diminished, and the mucosa assumes a nodular appearance sometimes described as cobblestone. The nodules may be polygonal in shape and their surfaces more reddened than the surrounding areas. In this form of gastritis, the symptoms may even more closely simulate ulcer pains; gas, belching, and weakness are commonly associated with hypertrophic gastritis. These are exemplified in the following:

Case 2. Gastric symptoms appeared as far back as April 6, 1934, when she complained of belching, epigastric distress, and lack of appetite. Laboratory reports were negative, as were x-rays of the gall-bladder. The gastric symptoms recurred intermittently, and on May 25, 1940, she was gastroscoped. The findings were as follows: on the midportion of the posterior wall of the stomach were patches of raised, biscuit-like projections with a hyperemic hue, separated by fine lines. The diagnosis of hypertrophic gastritis was evident.

Atrophic gastritis, in our experience, (bearing in mind that we are also examining a large number of patients with pulmonary tuberculosis) is the most common type. In this form the marked thinning of the mucosa is the outstanding feature. In the more advanced cases, arborizations of blood vessels may be seen through the thinned mucosa and there is frequently an almost complete obliteration of the rugae. The mucosa appears gray, greenish-gray, or whitish in color. The grayed areas may merge into areas where the mucosa appears thinned but reddish in color; the reddish areas probably represent the end phase of the initial acute stage of atrophic gastritis. In pernicious anemia and far advanced carcinomas of the stomach, the atrophic changes may be so diffuse as to involve the entire stomach. As to symptoms, the usual ones are moderate epigastric pain, anorexia where the weakness and loss of weight are pronounced, epigastric fullness, anemia, and nervousness. Examinations should be made at frequent intervals to detect the possible development of carcinoma.

Case 3. For six months before the gastroscopic examination, the patient had had weakness, pains in the abdomen, belching, and had lost 30 pounds in weight. He appeared markedly anemic. There was marked tenderness in the abdomen. The roentgenological examination was negative. The gastroscopy on May 17, 1940 showed that throughout the stomach was a thinning of and grayish cast to the mucosa, with areas where blood vessels were visible—a picture of atrophic gastritis.

Ulcers

IN THE diagnosis of gastric ulcers on the lesser curvature of the stomach, gastroscopy assumes a place secondary to roentgenology, particularly in view of the fact that benign ulcers, as has been mentioned before, on the lesser curvature of the antrum beyond the angulus are only rarely recognizable by gastroscopy. On the other hand, ulcers on the posterior wall and greater curvature are more frequently recognizable and differentiated from malignancies by gastroscopic examination. The following case record is of a patient who entered the hospital September 1, 1939.

Case 4. Upon admission, the patient had epigastric pain, hematemesis, and tarry stools of a few days duration. The bleeding gradually stopped. Ambulatory medical management failed to afford complete relief. X-rays in October, 1939, showed a negative stomach. He was gastroscoped February 16, 1940. In the first portion of the stomach on the posterior wall a benign ulcer was seen with evidence of a crater formation and a marked area of hyperemia around it. Swollen rugae were seen leading from the ulcer.

During the treatment of gastric ulcers, a more careful analysis of the progress of the healing of the ulcer can be seen with the gastroscope, since we have frequently found that lesions may appear to be healed and the deformity obliterated under x-ray visualization, yet still seen by the gastroscopic examination. In some cases where, both roentgenologically and gastroscopically, there is some doubt as to whether an ulcer be benign or malignant, it can be more closely watched by means of the gastroscope to see the response to therapeutic management.

The benign ulcer has usually a gray or yellowish white base with sharp edges. It is ordinarily round, but may be irregular, and the floor of the ulcer is depressed. The edges are usually on a level with the surrounding mucosa and not elevated as is ordinarily seen in a carcinoma. There is a halo of hyperemia around the active ulcer and the rugal folds are swollen, often leading from the ulcer in a cartwheel formation. The base may assume a brownish color if recent hemorrhage has occurred, or, in active hemorrhage, blood may actually be seen oozing from the crater.

Benign Tumors

BENIGN tumors, in our experience, have occurred in less than one percent of the cases gastroscoped. As a rule, no symptoms are produced unless the tumor is large enough to cause mechanical obstruction or severe hemorrhage. Occasionally the cauliflower-like papillomas may undergo malignant degeneration. If it is at all doubtful whether these papillomas be benign or malignant, they should be removed surgically. We have had one case to date in which the patient gave a history of a severe gastric hemorrhage and was found gastroscopically to have a benign polyp.

Case 5. The laboratory work on this patient gave no significant findings. X-rays showed a questionable filling defect in the pars cardia. During the gastroscopic examination March 29, 1940, a polyp was found high in the fundus near the cardia and was seen to bleed actively at the time of the examination.

Where benign polyps are found, they can as a rule be distinguished from malignancies by their smoothness and the lack of involvement of the surrounding mucosa. Frequently they are associated with atrophic gastritis.

Carcinomas

WHEN one considers the high percentage of malignancies occurring in patients past middle life which so often have only vague symptoms and in which the x-ray evidence is frequently not conclusive, gastroscopy is a welcome aid. Not infrequently can one make a positive diagnosis of a malignancy where roentgenologically no evidence of gastric deformity was noted.

Case 6. In one such case, the patient's history was of weakness and dyspnea for the past four months. There was marked secondary anemia, with a history of tarry stools, but the x-rays were negative. On May 10, 1940, the patient was examined gastroscopically. There was an extensive dirty gray membrane extending from the angulus to the pyloric sphincter on the lesser curvature side. The mucosa throughout was markedly thinned. The diagnosis was carcinoma of the antrum with atrophic gastritis. After these positive findings, the patient was re-x-rayed with special emphasis placed on the part where the lesion was seen and at this examination, the barium meal also showed a deformity at the pylorus.

Not infrequently lesions diagnosed as benign ulcers can be shown with equal positiveness by the gastroscope to be malignant. This may be illustrated by the following case gastroscoped May 25 of this year.

Case 7. Six weeks prior to the gastroscopic examination, the patient had a recurrence of an old gastric distress which started four years ago, at which time he had been diagnosed ulcer of the duodenum. Again the x-rays showed a duodenal deformity and also a very questionable deformity of the pylorus. The patient's response to medical management was not satisfactory. Gastroscopic examination was advised because of the possible deformity of the pylorus. The findings were as follows: there was a small ulcer near the pylorus in the antrum, about two-thirds of which was readily seen. The edges were elevated and purplish-red in color, and the crater was brownish-gray. The diagnosis of malignant ulcer was made and operation was advised. A diagnosis of benign ulcer was made from the gross examination of the lesion, but microscopic examination confirmed the gastroscopic diagnosis of malignancy.

The common malignant ulcer may be differentiated from the benign ulcer in that the edges are usually raised above the surrounding mucosa and form a thick wall with no evidence of infiltration to the surrounding tissue. In other words, there is a definite sharp line of demarcation between the ulcer and the surrounding mucosa. The wall is of a purplish-red hue. In the center is a crater, the base of which is a dirty brownish-gray, or it may be interspersed with green or violet tinges. The crater does not have the smooth punched out appearance as seen in the benign ulcer. In the far advanced type such a lesion may involve the entire antrum of the stomach.

Diffuse infiltrative carcinoma may involve the greater portion of the organ as seen in the leather-bottle stomach. The entire stomach may appear to be inelastic as evidenced by the frequent inability to inflate it with air. Occasionally only in inferential diagnosis may be made due to the fact that the stomach cannot be dilated sufficiently to separate the walls. The rigid, nodular, bulging surface is highly suggestive of a scirrhous carcinoma. The mucosa in these areas is usually paler than normal and sometimes almost gray in color. If ulcerations appear on the surface of such an area, the diagnosis is quite simple, especially if such areas have a tendency to bleed easily.

Polypoid carcinomas, as seen gastroscopically, have a well-defined base with nodules of varying sizes heaped upon this base. The mucosa surrounding the nodules may have the same orange-red color as normal mucosa or may be decidedly redder in color, similar to the shade seen in acute gastritis. This type of tumor may be as small as a hazel-nut or so large it is difficult to see with the gastroscope since the instrument may be pressing too close to the tumor to see it. The following case illustrated this type of malignancy.

Case 8. The patient had lost 26 pounds in five months. For six weeks before the gastroscopy, a lump in the epigastrium was noted. The stomach contents revealed about 20 cc. of reddish-brown fluid. The angulus and antrum were seen, but not the pyloric sphincter. About the level of the angulus there was a polypoid carcinoma with a reddish bleeding base. The midportion and upper portion of the stomach showed some atrophic changes, but no evidence of carcinoma. Operation was advised, and the resection also revealed a polypoid carcinoma of the antrum.

The majority of carcinomatous lesions are associated with atrophic gastritis. In reviewing the history of such cases of malignancies and atrophic gastritis, not infrequently the gastritis has existed for months and even years previous to the finding of a malignancy. Atrophic gastritis may, because of this almost constant finding, possibly be considered a forerunner of carcinoma of the stomach.

Gastric Hemorrhage

THE cause of hematemesis has been considered by most gastro-enterologists in the past to be due to chronic gastric or duodenal ulcer, benign or malignant, in spite of the fact that after the hemorrhage has subsided, careful examination failed to reveal any evidence of ulcers. Since the advent of gastroscopy, we often find that many conditions not recognizable by x-ray can cause massive hemorrhage. In the past, this group often came to surgery for relief, with probably 15 to 20% failures.

We now recognize many other conditions as common causes of gastric hemorrhage. Acute and chronic gastritis may frequently cause severe hemorrhage without any definite ulcers demonstrable gastroscopically or roentgenologically.

Case 9. This case illustrates the above stated point. The patient was brought into the clinic because of massive hemorrhage. Four weeks later the x-ray examination proved negative. The gastroscopic examination July 11, 1940, revealed the following: there were extensive areas of grayish-white mucosa with blood vessels plainly visible. This was a case of marked atrophic gastritis.

In a recent case of hematemesis which lasted for three days, gastroscopy revealed a thrombosed blood vessel which gave evidence of recent hemorrhage. This was the only positive finding, as the x-rays were negative. Another instance where the hemorrhage was caused by a gastritis, this time of the hypertrophic variety, is illustrated by the following example.

Case 10. The patient had had epigastric pain for four years, vomiting for several months, and had lost 35 pounds in one year. Physical examination was negative. No x-rays were done. The patient stated that upon several occasions he had had hematemesis. The gastroscopic examination June 7, 1940 revealed the following: there were marked hypertrophic changes throughout the stomach, beginning at the antrum and extending throughout. There were numerous nodulations of varying sizes and colors. Superimposed on this pattern there appeared to be an acute phase of edematous hemorrhagic areas. The diagnosis was diffuse chronic hypertrophic gastritis with diffuse acute hemorrhagic gastritis. After two months of medical management, the patient was regastroscoped and the acute stage had entirely subsided, leaving only a few patches of hypertrophic gastritis visible.

Conclusions

GASTROSCOPY is a diagnostic procedure in gastric disease that is an adjunct to, and not a replacement of, other methods.

2. It should be considered as an aid in diagnosing all gastric conditions which have not been definitely diagnosed by other methods and should be utilized in connection with positive roentgen and clinical diagnosis to confirm or add to the diagnosis.

3. It is of the utmost value in diagnosing the gastridities and of great worth in determining whether or not gastric lesions may be of a malignant nature.

4. Gastroscopy is also of value in diagnosing lesions not easily found roentgenologically.

Personnel in Industry

—*Selection and Maintenance*—

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AS THE time available will not permit any broad excursion into the many ramifications of my subject I am confining my remarks today to two topics which I believe will be of interest to an assembly of this character. They are:

1. The selection and maintenance of the airline pilot, and

2. The importance of visual efficiency in any industry. Both topics are closely related.

Vocational selection and industrial medicine are presenting new problems almost daily. Many of you have been actively engaged, either as teachers on the theoretical side, or as executives on the practical side in formulating a better system for the selection and maintenance of personnel in industry. More and more the progressive workers in this field are recognizing the importance of selecting the right man for the right job, based on a reciprocal analysis. This necessitates considerable survey work from a practical standpoint and the adoption of physical standards adequate to meet the requirements of each task under consideration. The airline personnel and medical workers are constantly engaged on such studies.

Some of the most important factors in a good personnel and medical program are the maintenance of a healthy, efficient, safe and satisfied group of workers. Each of these factors is of great importance to the other and each has a definite bearing on physical fitness. Without good health, an efficient job is difficult—and many times impossible.

Good maintenance on a bad motor is commendable—but good maintenance on good motor makes for near perfection in operating efficiency. This is true of people—and it is the constant goal of United Air Lines to select personnel who are good motors to begin with. Maintenance from a standpoint of health, safety, desirable working conditions, fair rates of pay and efficient supervision, all work hand in hand toward keeping our motors running smoothly.

Selection and Maintenance of the Airline Pilot

ALL airlines cooperate in the manufacture of aircraft by laying down certain specifications they would like to see embodied in the planes to be flown over their lines. When a new type of plane is produced, it is given a very careful service test and subjected to all the checks and double checks necessary to determine its air-worthiness and suitability for the transportation needs for which it was designed. Moreover, after it is accepted as a safe and adequate means of transportation, the plane, throughout its service, is subject to daily and other periodic inspections and check-ups, including a complete overhaul after not more

Read at Management Personnel Institute, Purdue University, May 1, 1940

than 500 hours of use, in order to assure that it is always in perfect working condition and absolutely safe from a performance standpoint.

Precisely the same rule is followed with reference to the pilots who operate the plane. Flying personnel are chosen with the same meticulous thought and care that applies to equipment, and after employment their physical well-being is always a matter of primary concern to the airline with which they become identified. Perhaps in no other calling does the old adage about a sound mind in a sound body have so much significance, if the interests of safety are to be adequately served. Since its infancy, and throughout its development and growth to its present size, United Air Lines has always been fully awake to the need for and value of a medical department, at least to the extent of having periodic examinations of pilots performed as a company measure for its own information and guidance, in addition to the physical examinations prescribed by the Federal government. It was not until three years ago, however, that United established an examining place or physical clearing house, of its own, headed by a full-time medical director. Other airlines have already taken or are now contemplating similar action. United's medical unit in Chicago is generally acclaimed by visiting physicians as the last word in what a modern and up-to-date airline examining center should be. It consists of 17 rooms and booths completely equipped with every diagnostic aid necessary in surveying and evaluating the physical fitness of an examinee.

The primary mission of any airline medical department is to conserve and promote the health of all employees, with special emphasis on the piloting personnel. This, United's medical department is attempting to accomplish by (1) aiding in the selection and enrollment for company service through properly conducted physical examinations of only those individual who are physically fit for the performance of the duties to devolve upon them; by (2) keeping such personnel in good physical condition through the application of modern principles of preventive medicine, and (3) in furnishing those who do become disabled with such counsel, advice and professional assistance as will tend to restore them to health and working efficiency.

Another and equally important mission is to conduct such observations and studies as will directly promote safety and comfort aloft.

United's medical director is responsible to and functions directly under the president. In an advisory capacity he also maintains close contact with other heads of departments regarding any part of their work which may have a medical aspect. He conducts research and experiments of his own connected with the promotion of safety and comfort, and maintains close touch with research activities elsewhere. He also exercises a broad sanitary and hygienic supervision over company activities to the end that a healthful environment may be assured all workers. Special precautions are taken to safeguard the health of the men who pilot aircraft. The causes and prevention of pilot fatigue are being constantly studied with a view to improving conditions in the cockpit and making work there more comfortable. All United pilots are required to use artificial oxygen whenever flying at any altitude above 10,000 feet, irrespective of their individual tolerance to partial oxygen deprivation.

Company physical examinations are of three classes:

1. Pre-employment, which are conducted in the case of *all* applicants for employment with United Air Lines. This policy is in keeping with the principle that safety will be promoted and accidents prevented if all personnel, ground as well as flying, are mentally alert and physically fit.

2. Periodic, which are conducted at prescribed intervals in the interest of health conservation. This examination assumes extreme importance in connection with the pilots and stewardesses who constitute our crews aloft.

3. Special, which are conducted as indicated by circumstances or when specifically ordered by the medical director. This examination plays a prominent role in all safety precautions taken regarding the physical fitness of all pilots.

A pilot returning to flying duty after an illness or injury is also given a special examination and is not allowed to resume flying until the company medical examiner reports him to be physically qualified.

The principal object of the periodic and special physical examinations conducted by the medical department of United Air Lines is the conservation and promotion of health. Therefore, the examination is thorough and in deviations from the normal all recognized diagnostic procedures are used in an effort to determine the character of incipient or obscure defects which will impair the health and usefulness of the individual examined. When such abnormalities are discovered the medical examiner makes suitable recommendations for the institution of corrective measures. It goes without saying that a pilot wants to keep himself physically fit.

Whenever I examine a pilot for the first time that he appears before me for a company examination, I invariably tell him that since he came to us physically fit, it is my job to keep him so if I possibly can, and that I can do so only by subjecting him to periodic physical examinations in an effort to detect and "nip in the bud" all deviations from the normal before they can become disabilities that might cut short his flying career. An approach like that usually elicits 100% cooperation on the part of the examinee, and many of them do require corrective measures from time to time. I derive no little satisfaction in being able to correct defects while they are still negligible, and thus keep a pilot fully qualified for the duties that devolve upon him aloft.

This doesn't mean that we do not encounter an occasional problem, which in the interests of safety, necessitates the permanent grounding of a flyer as the only reasonable solution. Like the rest of us, pilots are only human. In a laudable effort to keep physically fit they may, and in most

instances do, lead cleaner and better regulated lives than most of us. Nevertheless, in some cases, fortunately rather rare, there comes a time when they become the victims of a disability that cuts short their flying career. All airlines have similar cases, and I can assure you that it is a painful duty when a medical director has to take timely action that suddenly and irrevocably dislocates a man from his chosen career.

The high physical standards prescribed for flyers have been chiefly instrumental in keeping down the accident rate in aviation, but nevertheless it behooves us to remember that the supervision and checkups exercised by flight supervisors and other check pilots are of equal importance, and their efforts to weed out the unfit should meet with the same general approval as does similar action on the part of a medical examiner for strictly physical defects.

When medical examiners and operations executives keep a man on the ground, it is because they believe he would be unsafe in the air. When they let him fly, they stamp him as safe.

The country at large, and the flying industry in particular, need have no fear that our planes are not in the safest and ablest hands. In my opinion, better pilots could not be found anywhere.

The airlines of this country have just established a notable record of achievement. They have flown more miles than ever before without a single fatality in over a year of continuous operation. By keeping a watchful eye on the physical fitness of our flying and ground personnel we flight surgeons feel that we have contributed something to the attainment of this safety record, and the very happy state of affairs that prevails throughout the air transportation industry today.

It will be of interest to you to know that every applicant for employment with United Air Lines is put through identically the same examination as is a pilot. In building up a comprehensive health record on each employee we need all the physical findings that can possibly be brought out in a searching initial examination.

Now let us proceed with my second topic, which is, the importance of visual efficiency in any industry.

Importance of Visual Efficiency

IT IS quite generally recognized that the physical standards prescribed for flying personnel are the most exacting that could possibly be laid down for any vocation. The wisdom of this has never been questioned by anyone. And, moreover, we can point to the fact that we are still able to get candidates for flying training who can meet these high standards.

However, when we look at our physical yardstick rather critically we find that the barriers we have erected are found largely in the eye field. As a matter of fact, back in 1914 when the Army first drew up its physical standards for flying, the specifications regarding visual efficiency were so high, because of a strict adherence to the normal, that few, if any, of the candidates could pass the examination. This immediately resulted in a liberalization of some of the standards still within safe bounds.

The eye standards devised then are still in operation today. Instead of confining one's examination to a quick inspection, and a determination of visual acuity and color perception, a complete eye examination becomes a "stumbling block" to many flying aspirants. For example, in the U. S. Army during the World War approximately 100,000 applicants for flying training were examined by boards of especially trained examiners. Of that number, approximately 70% were qualified and 30% rejected, failed to meet the eye requirements.

Now, aviation is not the only industry in which good binocular single vision is extremely important. With its fast moving and intricate machinery, precision instruments, gauges, and what not, all keyed up to mass production, faulty vision certainly has no place in any of the other industries of today. In fact, because in many instances their visual requirements are just as exacting as those of aviation, I have often wondered why industry in general has not paid more attention to the importance of good eye performance on the part of their workers.

In many of the plants visited by me, the eye examination has been confined largely to a distant vision test with a Snellen chart and perhaps, a crude color test. Such procedures only scratch the surface as regards the real ocular requirements in any up and doing industry. This was brought home to me rather forcibly only a short time ago when I was privileged to visit and see something of the splendid work being conducted by the Kuhn Eye, Ear, Nose and Throat Clinic at Hammond, Indiana.

I was astonished by the amount of constructive work that had been carried on by them in the Calumet region, covering all types of industry, and now being focused on a specific research assignment.

Since I happen to know that Purdue University has played a prominent part in the study of production standards, abilities, and other knotty problems of industry and has made an invaluable contribution to the setting up of standards for physical aptitudes which in this instance are being correlated with visual aptitudes in a major training program I want to discuss briefly the findings, and then draw some conclusions as to the value of and need for, a continuation of such surveys.

The employees of 24 plants covering all types of industry and aggregating approximately 12,000 employees showed acuity defects of around 15%, even after correction, depth perception (stereopsis) defects of around 14%, color perception defects of around 6%, and muscle imbalances of around 25%.

All of these defects are important from the standpoint of visual efficiency. But among them the uncorrected visual acuity stands out as paramount and is of immediate importance. As the average of serious eye defects of all kinds varies between 8% and 15% throughout the district, it indicates a very appreciable group of workers that

need attention from the standpoint of their own comfort and efficiency, as well as from the standpoint of productivity and accident prevention in the plants in which they work.

As no other extensive industrial medical visual survey has ever been made elsewhere in this country to my knowledge, I do not know whether this survey is representative of conditions in industry generally. However, I am inclined to believe that it is.

Because too little recognition has heretofore been given to the fact that proficient visual performance can be secured only by a most searching type of examination, it is my hope that many of the industries in which such findings are important will widen the scope of their examination so as to include and acquire more exacting data in their admission standards.

The survey conducted in the Calumet area has been instrumental in standardizing a technique of visual testing and establishes now visual standards for industry which are both practical and scientifically sound.

Surveys such as these enable medical workers to point out existing weak-spots in personnel procurement.

They will permit of the accumulation of medical statistical data of such scope as to facilitate with reasonable accuracy the preparation of specifications on visual performance, or in other words, "Eyes for the Job." They will go far towards bringing about a comparative study of the demands on visual efficiency in various jobs in various industries, steel, soap, chemicals, machinery, offices, hosiery, etc.

But what are the advantages that can accrue to management in the compilation of factual data and other information through the medium of these large-scale visual surveys and revised admission standards? They are:

1. For the medical department—providing additional information on vision for compensation purposes, general and specific information on visual affections and needs of employees as a basis of rehabilitation policies.

2. For the personnel department — providing records for guidance and placement of employees, guidance as to desirable visual requirements for different departments and jobs, such as crane operators, inspectors, engineers, blue print workers, etc.

3. For the safety department—providing records for guidance as to minimum visual requirements for safety on various jobs and giving them clues to unsuspected occupational hazards.

4. For the production department — providing information on the relationship of visual efficiency to accuracy and productiveness of employees, with clues to detect impediments to production by reason of faulty visual performance, poor visibility, etc.

I have gone to some length in extolling the advantages as well as the wisdom of a more complete eye examination than is now given in most industries and I am practicing what I preach, even among our ground workers in aviation. I hope that my enthusiasm as a missionary in seeking converts to my way of thinking on this subject will be excused by those who do not see with me eye to eye.

My background of experience does prompt me, however, to hold firmly to the conviction that industry in general, in this fast moving age, cannot emphasize too strongly the importance and value of the physical examinations such as they have been conducting for years, but now bringing up to the same level the matter of satisfactory visual efficiency and the adaptability of "Eyes for the Job."

In closing, may I suggest that it behooves us and all others concerned in the promotion of industrial medicine to remember that among other considerations, our physical standards have the following objectives:

1. Long and efficient service.
2. Reduction of compensation, insurance and pension costs.
3. Reduction of absenteeism from sickness and injury.
4. Elevation of morale and increased efficiency.

Serious Conditions

—of the Eyes and Ears Following Minor Ailments—

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THE eyes and ears are substantial portions of the human anatomy that demand constant care and attention. The ever-pressing need of their use is essential in a large way to the preservation of the rest of the human body. They compose the two most important arms of defense in our waking hours. The ears listen to our approaching enemies and to our approaching friends. The eyes detect all visible objects, guard our stomachs and ward off all enemies of all parts of the body in all forms and in all spheres and walks of life. These two are the most essential of all five—and when every function of these two is gone man cannot live.

We may have to part with different parts of our sight apparatus. We may lose one eye and still see, we may lose one lobe of the brain and still see, and so on with the ear—as a matter of fact, we are providently blessed with sight even after considerable loss of many parts of our eyes. Again the same is true where we may lose a relatively large part of our hearing or our sight down to a minimum and still have a feasible communication with the outside world. We can lose one or the other of both and still hear and see. We can lose portions of one or the other of both and still hear and see.

Now with these facts in store we proceed with our subject relative to the pathology of injuries or ailments which lead to most serious conditions and

Presented at the Annual Meeting of the Association of Surgeons of the Southern Railway System, Charleston, South Carolina, June 6, 1940.

even calamities. Just like the tiniest pin prick on the skin may lead to sepsis and death, just so the least injury or contusion of the cornea may lead to ulceration and destruction of the cornea or a prick of the same may lead to a panophthalmitis and destruction of sight or more—to recapitulate, a lick or blow ever so light or the injury of a cinder may lead to either of the above cases of pathology. Also, the eyes may become diseased through many fields of allergy that may lead to loss of vision when minor things are neglected. A neglected cinder may lead to a corneal abscess, opaqueness and loss of vision. You may allow me here, though, to say that as to a cinder that the eye can stand a lot of punishment; and I have never in my practice seen trouble follow the removal of a foreign body from the cornea that was extracted within 48 hours, unless that person had a neighboring disease of the tonsils or sinuses.

Often the constitutional condition of a patient disturbed by allergic conditions of food or drink so lowers the protective forces of the body as to allow disease to take place through these avenues of entrance. Sometimes occupational diseases occur and through their neglect the eyes or ears become impaired and maimed.

Of course, we know that old underlying luetic conditions are perplexing and bring on plectynular ulcers or keratitis or atrophy of the nerve head, working gradually, but surely, to destroy the tissues and take away the sight. And right here allow me to say that a positive Wassermann is often absent where the diseases are located above the shoulders and it takes diligent search to placate the luetic condition in the blood of the patient.

Now the important factor I would suggest to be sure and aptly observe in small and tiny beginning complaints that may lead to loss and destruction, is an ardent seach for the underlying cause even though you hardly suspect the same. All these conditions that I have mentioned if taken in their very premature and pre-developing stages can be diverted and the functions saved and preserved.

Of course, in the case of allergy the cause of whatever nature, either local or constitutional or extogeneous, has to be sought. In many such cases the effects can be neutralized with local treatment until the cause is formed or the seasonal disturbance of whatever nature is past and gone. Sometimes we see a disease coming from a neighboring nasal condition which is affecting the eyes, and the removal of which relieves the patient. The sleeping quarters, the food, the clothing, or the atmospherical condition can be corrected in certain cases and the patient relieved. If you take the plectynular ulcers of children that lead to destructive ulceration and blindness, you will find that much can be done by observing the surroundings.

I once saw a school room which housed 60 children who were suffering from blepheritis. They were overcrowded, and were suffering from food deficiency that did not protect their eyes from the overcrowded condition and certain conditions of the midday rays of sunshine that were allergic. I advised the correction of the school room, the adding of the food deficiency, and after that they had no more trouble. They were able to overcome the actinic rays of the midday sunlight.

We discover in many cases that the proper nourishment is lacking or certain deficient vitamins need to be made up to carry on the certain exposure that is taking place. I have seen old people with ulcer of the cornea, in whom, on feeding them cod liver oil and also instilling cod liver oil in their eye along with an intermittent instillation of some mild antiseptic, recovery rapidly took place. I have seen cases where the removal of a spark was instituted under non-aseptic procedure and seemingly a hematogenous infection ensued, followed by a cloudy vitrous and pamophthalmitis. This, perhaps, was a lack of cleanliness.

NOW I take you around to the ear, a most important communique between man and his outside world. The powers of the rays of light convey color along with form by the protracted vibrations. Vibrations convey expressions of sound so vitally needed to detect our friend or enemy.

It is a fact that a C^2 fork will be heard in the ear of one person for only 35 seconds and by another for 65 seconds. Also, one person may hear well for a C^2 fork and very deficiently for a C^4 fork, and vice versa.

For the proper function of the ear, vibrations should always be appreciated at any rate. The conditions that interfere sometimes come along slowly and unknown to the patient. Considering countless circumstances we are brought face to face with the fact that any interference with ventilation of the ear causes loss of hearing. We promote usually adequate assistance by promoting drainage and ventilation.

We have local conditions and constitutional—such diseases as scarlet fever and luetic conditions are sometimes very rapid and destructive. Local conditions include catarrahal conditions of the middle ear and fungus conditions of the outer ear and then, too, the adenoids of children. Sinus diseases and tubal catarrh often contribute to the undoing of the functions of the middle ear and lead slowly to different stages of deafness. It is often found that certain allergic foods, or even certain pathology of the stomach, cause upper respiratory disturbances and indirectly interfere with the function of hearing inducing certain and varied stages of deafness. We find certain foods that cause eczema of the outer ear and with that certain moisture and temperature a fungus growth comes as complacently as the proverbial mistletoe of yuletide days, but is not so bewitching as those enchanting twigs. It develops, and eventually becomes extremely hard to control and cure. I have found no better remedy which is practically specific than pure grain alcohol and boric acid locally. The perturbing foods and any other causes are to be left off. I have seen patients who eat freely of cheese relieved of tinnitus when told to

quit, and yet I find that the eating of butter is one of the best foods in treating any patient with ear disease.

IN CONCLUSION I want to stress the fact that many conditions of the eye and ear fail to respond to our treatment because we fail to consider some constitutional dyscrasia or underlying cause. The blood of the patient needs to be examined or the allergic test needs to be exemplified in addition to all the other things we are doing. Sometimes an excursion around a patients living quarters will reveal a field of wild onions or a vast pasture of Bermuda grass; or it may be found that chickens are eating Bermuda grass and the patient eating the eggs, creating an allergic condition. Sometimes the patient is drinking the milk from cows that are feeding on Bermuda grass. This needs to be stopped.

In eye conditions some patients need vitamin A and Ruboflavin and in ear conditions some patients need vitamin B.

One of the facts I want to stress is that practically all conditions of the eye and ear outside of injuries are secondary. The cure may need attention *per se*, but the patient needs to have the primary cause relieved. The decay of a molar tooth will sometimes cause an ulcer of the cornea in the outer lower quadrant. The lack of the intake of certain vitamins will allow the development of a cataractous crystaline lens. It has been rumored that an abnormal supply of blood sugar will cause a glaucoma. We feel that keeping a perfect opsonic index and the body at par is paramount.

In the case of the ear, hardly a condition ever exists that does not prove itself secondary to some disease of the upper respiratory tract or to some existing allergy which is extogenous, hematogenous or otherwise. On the whole, the primary pathological conditions of the eye and ear are few and due to some exposure from without or within.

Lead Poisoning

—Criteria for Diagnosis—

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THE diagnosis of lead poisoning cannot be made upon any one criterion or finding. This paper therefore will not be an enumeration of laboratory criteria, though these make up an important part of the complete diagnosis. It will not present only a clinical picture either, but an integration of both.

One cannot specify diagnostic criteria without defining the great difference between lead absorption and lead intoxication. Lead absorption is not lead poisoning, though many have loosely regarded it so. Laboratory findings, such as determination of lead in urine or blood, are mere evidences of lead absorption. There is no arbitrary value of lead absorption which of itself spells lead poisoning. I know of no single laboratory determination which can be substituted for clinical experience.

It is essential therefore that we be entirely clear in our minds as to what we mean by the phrase "lead poisoning," both acute and chronic. Lead absorption simply means what it says, that lead has been absorbed into the body economy, not simply ingested and passed through the gastro-intestinal tract, as occurs with much of ingested lead. When we find lead in the blood or in the urine, we know, of course, that this lead has been absorbed directly into the system and that it is circulating in the blood stream and is distributed into the other tissues of the body, including the bones, as well as being excreted through stool and urine.

Lead intoxication is lead poisoning. As Paul Reznikoff[1] has so aptly said, "Lead poisoning is present when the subject has colic, palsy, encephalopathy or anemia." Lead intoxication presupposes an existing lead absorption, so we must spend considerable time evaluating this basic problem of lead absorption as such. This is a definite condition not to be confused with lead intoxication or lead poisoning. Lead absorption in moderate amounts is not necessarily harmful and in many instances is non-disabling. When discovered lead absorption should be treated by reduction of further exposure with proper protective engineering devices, in order to prevent the development of a real lead intoxication.

Lead absorption is indicated on physical examination by the finding of a lead line in the gums opposite carious teeth or in gums inflamed with pyorrhea. Such examination must be made with the use of a lens under proper illumination. If present the lead line will stand out as a distinct row of blue dots directly in the gum tissue near the tooth margin. The absence of a lead line in a mouth with good oral hygiene does not, however, rule out lead absorption. On the other hand, the absence of a lead line in a mouth with marked oral sepsis usually indicates that there is no clinically serious amount of circulating lead.

Lead absorption is also indicated by laboratory studies, as in the finding of lead in the urine. It is to be remembered, however, that even in normal individuals, with no occupational lead exposure, lead may be found in the urine in amounts of .01 to .08 mgs. per liter with occasional values as high as .10 and a mean value of approximately .035 mgs. There has been a quite general agreement upon this subject among men of experience such as Kehoe[2] of this country and Tompsett of England.[3] I am aware, of course, that there is still some difference of opinion as to the value of different methods for the determination of lead in the urine, whether it be some modification of the chromate method of Fairhall, some type of dithizone method or, a spectrographic method. While the first method does not show quite all the lead and some modifications of the dithizone method may occasionally show more than the actual amount of lead, because of the

Presented at the Twenty-Fifth Annual Meeting of the American Association of Industrial Physicians and Surgeons, New York, June 7, 1940.

interference of other metals or too wide variations in the blank, the spectrographic test, which theoretically is the most accurate, is very difficult to apply in routine work. In any event, in the hands of experienced operators, the differences between these methods are too small to concern clinicians.

Urinary findings are really of greater clinical significance than those of the blood because, as Kehoe[4] has shown ("Lead Absorption versus Lead Poisoning"—a paper as yet unpublished) the urinary response to lead absorption is both more prompt and more definite than the blood. The urine determination is also less likely to be altered by errors in technique, both in obtaining the sample and in analyzing it. However, blood lead values usually correspond with urine lead values so that for clinical purposes the much simpler urine lead determination is sufficient. In doubtful cases we should recall, however, that in the normal individual blood lead values may range from .01 to .06 per 100 gms. of whole blood with an occasional value as high as .07 and with a mean value of approximately .03.[2,5] The safe level of blood lead which is compatible with a non-hazardous continuation of a given lead exposure has not as yet been adequately determined. Because lead workers who do not have symptoms are not forcibly brought to the attending doctor's attention, the high blood and urine levels which are consistent with good health in lead workers over a period of many years have not been published in any great detail, as yet. However, as men of broad experience with lead absorption gradually compare their clinical and laboratory findings, it is becoming apparent that surprisingly high levels of blood and urine lead have occurred in the healthy lead worker with no suggestion of lead poisoning.

Another important sign of lead absorption, which some laboratory workers may tend to overlook, is the time honored study of stippled red cells in the blood smear. It is, however, an important measure especially because it involves the technique with which all physicians are familiar, namely the Wright stain. While some men feel that such polychromatophilia can be demonstrated more rapidly by other staining modifications such as Manson's or methylene blue methods, or even by the basophilic aggregation method, the most practical method for general use, according to my experience over the past 15 years, is still the demonstration of stippled cells by the Wright stain. For purposes of comparison the stippled cells should be counted in 50 oil immersion fields.

It is admitted that stippled cells are not in themselves diagnostic of lead poisoning any more than any other one laboratory determination. One may find stippled cells with any advanced anemia such as may occur with benzol poisoning, primary anemia or Hodgkin's disease. However, the point of characteristic importance with the stippled cell of lead absorption is that it appears first without an associated anemia, rather than late after the development of an anemia as is usually the case in non-lead pathology. The so-called normal range of stippled cells, moreover, which may be found in any individual, regardless of anemia or lead absorption or other pathology, is from one to 12 per 50 oil immersion fields or from 100 to 1000 per million red blood cells, according to the European standards. Kehoe also gives a mean value of 340 per million erythrocytes[6] for 784 healthy persons with no history of occupational lead exposure. When one finds, however, more than 12 stippled cells per 50 fields examined, or more than 1000 per million red cells, without an associated anemia and in the face of known lead exposure, the stippling is often the result of abnormal lead absorption. Finally, in the study of stippled cells, one must emphasize again that it is not a single determination which is of diagnostic importance but a serial study of the "stippled cell curve."[7] This is built up by repeating the stippled cell count every one to two days where lead intoxication is suspected and certainly once in two weeks in any instance of known heavy lead exposure even in the absence of symptoms. The significance of a characteristic stippled cell curve will be developed further when I discuss actual lead intoxication.

The point at which lead absorption becomes lead intoxication is characterized by the development of illness. This may arise from a grossly increased lead exposure or from certain physical or metabolic changes in the individual, such as acute infection or acute illness of any type, alcoholism, excessive fatigue and perhaps many other factors. Then it is that the lead worker is disabled and that is what we call lead intoxication. With the first sign of acute infection there may still be opportunity to recognize the latent impending lead intoxication by the careful correlation of the stippled cell curve and the clinical symptoms of slight anorexia, constipation and shifting abdominal cramps in time to prevent a really disabling lead intoxication.

While lead absorption is of itself not necessarily harmful or disabling, there are three main types of lead intoxication which are temporarily harmful and certainly disabling though for different periods of time. The most common type of true lead intoxication or acute lead poisoning is the abdominal type called "lead colic." Approximately 90% of the cases of acute lead poisoning with which a physician comes in contact are of this type. This is manifested by the typical picture of constipation followed rather rapidly, in a few days, by complete constipation or obstipation, associated with the violent abdominal pain which we call lead colic. Following excessive lead exposure with resultant lead absorption, this lead colic intoxication always shows association with abnormal quantities of lead in the urine and blood; the elevation is not always great, especially in the case of the blood. However, the lead colic type of lead intoxication is always associated with a characteristic rise in the stippled cell curve as seen on a blood smear, if smears are taken immediately at the onset of symptoms.

In fact, while there has usually been an ade-

quate warning in the gradual rise of the stippled cell curve for several weeks before the onset of clinical lead colic, there is with its actual precipitation a sudden rise of from 20, 40, 80, 100, 400 or even 1200 stippled cells per 50 fields. This spike-like curve frequently develops within one to three days after the onset of symptoms. Usually there has been no premonitory drop in hemoglobin, but with the onset of disabling lead colic and the marked rise in stippled cells, the hemoglobin drops rapidly from 80 or 90 down to a level of 40 or 60%. This sequence of events makes it essential that every physician in industry pay more attention to his stippled cell curves than to hemoglobin readings, and certainly that he must not wait for the drop of hemoglobin before instituting proper prophylactic and therapeutic measures. This I have discussed elsewhere.[7] With the lead colic type of lead intoxication there is immediate disability but this is usually temporary with such an improvement of symptoms and even anemia that the individual is able to return to work in four to eight weeks, most commonly with no residual effects.

Cases of lead palsy, a second type of true lead intoxication, occur relatively rarely. They usually show an associated history of preceding neglected lead intoxication of the abdominal colic type, associated with typical lead constipation and characteristic stippled cell curves. With the onset of the actual palsy, the stippled cell level may not be excessive and often not diagnostic of itself. The healing period may extend from two months to two years. Even then any residual disability is rare. Lead neuritis is motor rather than sensory and involves extensors rather than flexors.

The third type of lead intoxication, manifests itself in the stupor or in the violent delirium of lead encephalopathy or brain involvement. These cases are in a class by themselves and may not show the warning of previous lead colic or stippled cell rise. Their history is a characteristic story of overwhelming lead exposure seen most dramatically in the case of exposure to tetraethyl lead, with sudden onset of irritability followed in rapid succession by marked excitability, mania, coma, and frequently death. Less dramatic types are characterized by heavy lead exposure with headache, dizziness, confusion and somnolence as the predominant picture. Such cases, usually marked by convulsions at the onset, are not uncommon in children where the lead seems to have a prediliction for growing nerve tissue. These cases are uncommon in modern industry, where we are concerned with adults. With the ordinary precautions now widely used against excessive lead absorption, lead encephalopathy has largely vanished from American industry.[8] There is an interesting observation to be made here even in this most intense type of lead intoxication, the only type that kills, that providing the patient has survived the acute stage, after a healing period of disability of two months to two years, there may be no residual effects. In children, however, residual damage is not uncommon.

All of these three types of lead poisoning are acute and only these three types should be dignified by the term lead poisoning. I do not believe that there is any such thing as chronic lead poisoning though on the other hand, there is, of course, chronic lead absorption. What has been erroneously called chronic lead poisoning, when investigated, usually turns out to be a series of major or minor episodes of the three types of acute lead intoxication enumerated above. Each episode is a distinct and new lead intoxication. It is only after an acute and observable lead colic that pallor, anemia, and weakness develop. A patient cannot drift into these symptoms without first having had a real lead intoxication in which the blood has been characteristically affected with stippled cell rise and later a drop in the hemoglobin.

Chronic lead poisoning is a misnomer when applied to the assumed untoward effects of continued lead absorption on the lungs, stomach, kidneys, liver, arteries, and vasomotor apparatus of adults. High levels of lead in blood or urine are not diagnostic of chronic lead poisoning. Anyone with considerable experience observes numbers of exposed persons who have gone on for years with urine lead levels ranging from 0.10 to as high as 0.20 mg. per liter indicative of unusual lead absorption and yet who have never showed evidence of clinical lead poisoning. They are simply examples of normal lead metabolism, that is, absorption and excretion without symptoms or damage. This normal lead metabolism is a proved fact within certain limits of intake and output.[9] The body can handle the continued absorption, storage and excretion of small amounts of lead with no damage at all just as it can handle other drugs in proper proportion. In other words, the lead is of itself not an immediate tissue poison and its deleterious effects depend upon its concentration in the circulating blood stream. While lead is being absorbed through the respiratory tract, it does not damage that organ locally, neither does it become concentrated in the local lymphatic depots which drain the points of absorption because it is soluble in the body fluids and its concentration thus reduced by circulation. It does not set up any fibrosis in and about such structures as does occur in the case of silica inhalation. Even in the most concentrated sources of lead exposure, as in the lead smelters, paint grinding trades or storage battery industry, there is no increase in tuberculosis.*

As the lead circulates through the organs in the amounts now experienced in ordinary industry, there is no apparent damage to the adult lungs, liver, or kidneys.[9-a] Even in the great excess of lead circulating in the blood in the semi-experimental cases of cancer treated by colloidal lead injections there has been no characteristic demonstrable damage to the kidneys, such as is found in the development of a nephritis.

Wagener and Kieth,[10] in reporting some of their Mayo experiences, state "acute renal insufficiency

* According to DR. ALICE HAMILTON in Industrial Toxicology.

rarely occurred when acute lead intoxication developed in cases of cancer in which large therapeutic doses of lead were given intravenously." It should be noted, of course, that in this discussion we are speaking entirely of adults in industry and are not concerned at all with changes developed during childhood and adolescence, such as have been reported and attributed to the effects of lead absorption by Nye.

There is no proved increase of ulcer of the stomach moreover, or high blood pressure, in cases of increased lead absorption. There are no modern figures in any long time study which show a consistent increase in blood pressure or arterial changes in any greater amount than could be expected in the gradual increase of degenerative changes in any group of advancing years, regardless of occupation.[11-12]

The blood forming centers in the bone moreover are not damaged by the deposits of the lead therein. The fact that lead is excreted both through the bile and thence into the stool as well as by the kidneys, serves amply to safeguard any dangerous amount of circulating lead in the ordinary cases of lead absorption. All these instances of lack of evidence of visceral pathology in chronic lead absorption simply proved clinically what has been shown experimentally, that there is a normal lead metabolism. After absence from lead exposure for one or two years, lead intoxication practically never develops. Following cessation of lead exposure the concentration of circulating lead becomes rapidly diminished. There is considerable evidence to show that the recently absorbed lead is deposited first on the trabeculae of the bones and thence into a much more stable combination in the solid portion of the bones where it is covered further by subsequent deposits of calcium. This accounts for the fact that though an individual may excrete small amounts of lead in urine and stool for an indefinite period following the absence from lead exposure, he rarely develops subsequent lead intoxication after one or two years. The fact, then, that the body is able to handle small amounts of circulating lead both in intake, storage, and excretion, all show that there is a normal lead metabolism without symptoms or appreciable bodily damage.

Just where normal lead metabolism is deranged so that the body develops disabling symptoms is difficult to predict or to measure by any one laboratory finding such as by a blood lead determination. Even recent authors[13] who believe that they can diagnose clinical lead poisoning from blood levels of .1 to .2 mgs. per 100 cc. of whole blood, often hasten on to say: "As has been the experience of other workers in this field, we have been unable to find any direct correlation between the level of lead in the blood and the severity of the symptoms." This admission of lack of any exact correlation between blood lead and the clinical picture corresponds exactly with the experience of many of us of a similar lack of correlation between the urine lead values and the clinical picture.[14] This lack of definite correlation between the signs of absorption of lead found in blood and urine and the clinical findings is a marked contrast with the usually exact correlation between the rising stipple cell curve and an imminent lead intoxication, lead colic type. This, as I have said, is the predominating type of lead poisoning actually met with in industry, so that the clinical history and the stippled cell curves are of the greatest practical significance in the actual diagnosis and control of lead intoxication in industry.

A persistent finding of .15 to .2 mg. of lead per liter of urine does not necessarily point to lead poisoning in a given case but it does mean that there is a potentially dangerous condition of lead absorption which calls for correction.

Whether a man is to show evidence of lead poisoning does not depend on his signs of lead absorption as shown in the gums, blood, or urine, but simply on his ability to keep pace with the excess of absorbed lead by safe distribution or excretion. If he cannot keep pace and if no physician is in charge to anticipate an imminent lead intoxication, the worker may well be subject to acute attacks of lead colic, the rather rare lead palsy, and the extremely rare lead encephalopathy. The deciding factor is the severity of the exposure. The deciding criterion for diagnosis is the effect of this degree of lead exposure on the whole individual human adult shown by observable changes in the blood picture of the stippled cell curves associated with a characteristic syndrome of lead obstipation and colic followed rarely by palsy or encephalopathy.

In any event, criteria for the diagnosis of lead poisoning cannot rest upon any one finding of lead absorption in blood or urine, but must continue to be based upon the whole picture of known lead exposure together with objective evidence of lead absorption as well as upon typical signs and symptoms of lead intoxication, all viewed in the light of clinical experience.

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An Analysis of 1000 Deaths

— *Among Railroad Employees Subject to Periodic Examination* —

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WE HAVE been interested in the cause of death among our employees since we were appointed to our present position in 1936. In the spring of 1937 we began keeping records of all deaths among employees subject to periodic examination. At our annual meeting in September, 1937, I presented a paper which analyzed 184 deaths. We now have records of about 1250 employees. Between 300 and 400 die each year.

We have had some difficulty in compiling these records because of the trouble in getting accurate cause of death. Our source of information in most cases has been the superintendent's office. In the beginning very frequently the reports were received stating that the employee died from natural causes, or from such causes as heart trouble, kidney trouble, stomach trouble, liver trouble, etc., all of which were in many cases of very little use to us. We have written many letters in an effort to obtain the true cause of death, and there has been great improvement in this respect. We have hopes that eventually we may be furnished in all cases with the cause of death as shown by the death certificate. The causes of death as given in this list were obtained by a comparison of the report received from the superintendent with our records. Let us say that the best information we could obtain indicated that the employee died from heart trouble. If, from the records in the file, the actual diseased condition could be evaluated, the cause of death was taken from the file. If, on the other hand, the record showed no indication of cardiac or circulatory abnormality, the cause of death was stated to be unknown. However, if it was learned that the employee had died suddenly from a so-called heart attack, the cause of death was put down as coronary occlusion regardless of the office records in the case. As far as possible the actual disease causing death is named in this report. For example, if the report indicated that the employee died of gangrene of the leg and it was known that he was a diabetic, the cause of death is given as diabetes. Another example is pneumonia. Where it was obvious that pneumonia was simply a terminal condition, the underlying disease is given as the cause of death.

With the exception of coronary occlusions, all circulatory diseases are grouped in one division. This includes valvular heart disease, both luetic and rheumatic, cardiovascular renal disease, arteriosclerosis, myocarditis and cerebral hemorrhage. Coronary occlusions are placed in a separate class, although many of them occurred in employees known to be suffering from vascular diseases of various types.

The fatally injured are separated into two divisions; viz., those injured in line of duty and those not injured in line of duty. There is no assurance that this division is accurate. Many of the reports indicated definitely that the employee was injured while on duty, and others indicated that he was killed in an automobile accident or otherwise. Many of the reports, however, merely stated that the employee was fatally injured without any additional facts. These are all classed as being injured not in line of duty. Several employees died following injury but were not placed in the classification of fatally injured because it was felt that the injury was merely contributory to the death, and that the true cause of death was pre-existing disease. One hundred and eight employees in this list were fatally injured; 72 not in line of duty and 36 in line of duty. Four

CAUSES OF DEATH

Vascular Disease excluding Coronary Thrombosis		255
Coronary Thrombosis		220
Neoplastic Disease		89
Fatally Injured	72	
Fatally Injured L.O.D.	36	133
Drowned	4	
Suicide	21	
Pneumonia		62
Pulmonary Tuberculosis		26
Peptic Ulcer		20
Diabetes		15
Chronic Nephritis		15
Appendicitis		13
Brain Tumor		7
Cholecystitis and Cholelithiasis		7
Meningitis		7
C.N.S. Lues		6
Bacteremia		5
Cirrhosis of Liver		5
Prostate Hypertrophy		5
Intestional Obstruction		4
Bronchial Asthma		4
Leukemia		3
Hodgkin's Disease		3
Miscellaneous		26
Unknown		70
Total		1,000

Engineers and Firemen	284	1,000
Other Employees	716	

Average Age at Death	55.1
Average Age of Engineers and Firemen	55.38
Average Age of Other Employees	55.06
Deaths resulting from Vascular Disease	47.5%
Engineers and Firemen	47.0%
Other Employees	47.6%
Deaths from Coronary Occlusion—220	22.0%
Engineers and Firemen—63	22.1%
Other Employees—157	21.9%

Presented at the Seventeenth Annual Meeting of the ASSOCIATION OF SURGEONS OF THE NEW YORK CENTRAL SYSTEM, New York, September 24, 1940.

were drowned and 21 committed suicide. It is quite possible that the unstable business conditions of recent years contributed to the number of suicides. It was a surprising revelation to us that more than 13% of the employees in this list died of a violent death.

The average age at death for the entire group was 55.1. Of the 1000 employees listed, 284 were engineers and firemen. The average age of this group was 55.38 years. There were 716 employees in other branches of the service, the average age of this group being 55.06. This seems to be a very small percentage in favor of engineers, yet in a group of 1000 men this represents 300 years of life.

The figures on vascular disease are of interest. The statement has been made that this type of disease is more frequent among engineers than among other railroad employees; in fact, it has been claimed that heart disease is an occupational disease of engineers. Whether such a statement is based on any statistical record, we do not know. Such statements are not borne out by the figures shown in this list. There was a total of 475 deaths from vascular diseases of all kinds—47.5% Forty-seven per cent of the engineers died from this cause, and 47.6% in the remaining group. We believe that in any group whose average age is 55.1 approximately 50% die from vascular disease, and it is probable that among those who died from unknown causes there were sufficient deaths due to vascular disease to bring the percentage to 50% in this group.

There were 220 coronary deaths. Sixty-three of these were engineers; 157 in the remaining group. In the entire group 22% died from this cause. The percentage of engineers was 22.1, of the others, 21.9%. In other words, the percentage of coronary deaths among engineers and those not engineers was almost exactly alike.

It will be noted that the cause of death is listed as unknown in 70 cases. In most of these a cause of death was furnished which could not be verified by the record and was not sufficiently specific to be included in a list of causes.

Industrial Medicine

—*As a Specialty*—

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I BELIEVE Industrial Medicine is a specialty, one for which there is a very definite need in the world today. How to best prepare for it is a question of vital importance to many a young physician, as he tries to read his future in the hope of finding out just what the rewards in medicine are, before he enters a given field.

Sometimes a backward glance is helpful, for from the pages of history it is often possible to foretell the possibilities which lie ahead. To begin with, may I explain that this paper represents no one's opinions but my own. Last year, when you were good enough to invite me as an official representative of our American Association, I could not, perhaps, have spoken so frankly. If what I have to say seems to apply more to the full-time practice of industrial medicine in other sections of the country, it is simply because that is the only aspect of our specialty that I know at first hand. Nevertheless, I am no stranger to your part of the country, for I have taught in two of your medical schools. As a matter of fact, I went from a full-time job of teaching, to a full-time job in industry.

Because I was born and raised south of the Mason-Dixon Line, I am a Southerner at heart. Both the Woodys and the McIvers all come from Virginia and the Carolinas, and I, myself, was born and raised in Louisville, Kentucky. Perhaps that is why I have such unqualified admiration for the industrial development that has taken place in the southern states during the past generation. Anyone who knew the Piedmont 30 years, ago, cannot forget the contrast with conditions as they are today. In spite of all the problems that have arisen in the last few years, I cannot help but feel that this is only the beginning, and that the Seaboard States are, industrially speaking, the main hope of our nation.

Material progress will of course, give rise to many difficult situations, some acute, some chronic. The problems of health arising within industry are quite distinct from those of the private patient and the private practitioner. The physician in industry must be trained for his job, and as yet a standard course of training is still unformed, still unofficial. But adequate preparation for the practice of our specialty is becoming more and more important, for now that industry is beginning to see the need for medicine in its factories and offices, we must hold on to that which is good in old-fashioned medicine, even as we attempt to shoulder the responsibilities which are fast presenting themselves for solution.

Questions present themselves thick and fast. It would take an expert from every field to give comprehensive answers, but they are well worth thinking about, and I present them to you in the hope that ideas and solutions may be forthcoming. Is industrial medicine really a specialty? Is there a need for it? Emphatically, we all say: "Yes," but as you all know, many important and able physicians disagree with us. How can we best keep abreast of all the new specialties which the industrial man should know, and yet remain specialists? How can we best keep in touch with hospitals and private practitioners that we may work hand in hand with them, each supporting the other, particularly in industrial speed-ups and national crises.

I do not believe that specialism is an evil, for no human mind can compass the entirety of our present-day medical knowledge. In the church, one pastor may suffice to guide his flock through this life, in courts of justice, one lawyer may be able

Presented at the Third Annual Meeting of the Georgia Association of Industrial Surgery, Atlanta, September 25, 1940.

to look after the best interests of his clients without outside help. Even in such an expanding profession as engineering one will occasionally find a mechanical engineer in charge of an oil refinery or a chemical engineer as the head of a manufacturing concern. But we doctors cannot do that. An ophthalmologist cannot be the head of an obstetrical service, the best pediatrician cannot undertake surgery of the brain, the most skillful radiologist cannot possibly portray the intricacies of the human mind on his sensitive film.

Not only in medicine, but in all professions, is the pattern changing. The brilliant preacher has an assistant who makes social calls, does the office work, is pleasant and present at all the female functions where flutter and gabble take the place of serious attention to religion. The lawyer trained in property and contracts turns divorce cases over to the man who is effective before a jury. Even in the profession of engineering there are at least half a dozen sub-divisions. Just so the pattern in medicine is beginning to take shape, lines of demarcation are becoming more definite, more rigid, as indeed they ought. It is coming about by a sort of mitotic cell-division. Indeed, differentiation into separate specialities is taking place before our eyes.

Medicine, in its broader sense, has ceased its ameodoid movements; here and there is protoplasm seems to collect around some special interest, some new technique; and soon, a new specialty buds off as a separate cell, distinct, and yet how closely it does really depend on the rest.

The analogy might be carried on one step further. One might describe the atrophy and reabsorption of cells that once had a proud and independent existence. We no longer hear of societies of microscopists, of electro-therapeutists, and the climatologists are represented by but one national organization. This is all as it should be. Specialties should be re-absorbed once they have served their purpose. Otherwise we should be forced into the situation which Herodotus described over 2000 years ago when he wrote this of the medical profession in Egypt:

"The art of medicine is divided amongst them, each physician applies himself to one disease only, and not more. All places abound in physicians, some physicians are for the eyes, others for the head, others for the teeth, others for the parts about the belly, and others for intestinal diseases."

Contrast this hard and fast formalism, with that famous aphorism of Hippocrates which begins, "Life is short, the Art long, opportunity fleeting, experience treacherous, judgement difficult," and you will have little doubt as to which civilization was producing the better doctors. Perhaps Chaucer more nearly suggests our own feeling when we are confronted with so much that we would like to learn but that there is no time for, in the press of daily duties. He says so simply, "the Lyfe so short, the craft so long to learn!"

Because medicine is progressing so rapidly, what with new discoveries, new drugs, new methods, specialism is absolutely necessary, and it is indeed a privilege to belong to a profession which is going ahead, not standing still. The dictionaries give two definitions for specialist. First, "one who limits himself to a particular branch of a profession;" second, "one who has a special knowledge of a particular subject." One is negative, one positive. I prefer the positive one, the broader definition, which has scope and promise. And yet one of the recently organized American Boards will not admit a candidate to its examination until he gives assurance that he limits his practice to that specialty and that he will continue to do so. I for one believe that the only satisfactory way to solve this dilemma is to emphasize what a man knows, for after all, the more he knows, the better for his patients.

To be sure it is really only during the last 20 years that we have had men who limited themselves entirely to industrial medicine. As yet the time has not come when we can define a specialist in terms of what he will not do. Actually perhaps the best definition of a specialist in industrial medicine is—one who possesses knowledge of the medical problems that are constantly arising within manufacturing and business organizations.

NOW, how to obtain the very special skill necessary to the practice of medicine in a great manufacturing organization is quite another thing, and one which may be answered in many different ways. Up to now each industrial physician has, perforce, blazed a trail of his own, to his job, rather than followed along the paths of others. Perhaps one of the most interesting ways to find out how a man became an industrial physician, is to trace the course and careers of a few of the present leaders in our American Association. Twenty-five years ago there were no graduate courses in industrial hygiene. A man got his preparation in other specialties and usually entered industry because of his great interest in some special problem, and went there because of the opportunity for him to study that problem at first hand.

Loyal Shoudy, for instance, got his training in surgery with Dever, Bill Sawyer was with Frazier, Clarence Selby was Crile's assistant. Cassius Watson taught gynecology before organizing the medical department of the American Tel. and Tel. In public health Clarence Selby's name appears again, and R. R. Sayers still belongs to the Public Health Service though he is now Director of the Bureau of Mines. Lloyd Noland got his training in the army, while Dan Lynch was one of the few who actually grew up in his own job. The list could be longer, you yourselves, no doubt have been conjuring up names of friends, as I speak; but the background of these men shows very clearly the value of a broad and diverse professional background.

Now, the pioneer days of industrial medicine have passed; no longer can the industrial physician just grow, like Topsy. The question is, how best can we train the medical graduate of the present day? How can we get him into industrial practice with enough training to cope with his problems before he is 30 years old? Is it possible

to give him enough training in surgery for him to be able to treat industrial injuries adequately? Enough training is internal medicine to prevent occupational disease? Enough knowledge of psychiatry and neurology to handle the cases of maladjustment he will most certainly meet?

The answer to that question is distinctly a difficult one, for it would seem to prove that leaders of our specialty must have a peculiar catholicity of training, that one year's apprenticeship is not enough to give a medical student the broad understanding of the human mind and body which is necessary to make a successful plant physician. Perhaps it is well that most of the plants in the United States have less than 250 employees and that the doctor is not on full time, for it permits a fine flexibility for a doctor to care for his own private patients, and to care for those of industry in the same day. One practice can supplement the other to the benefit of both, and no doubt the big manufacturing plants will learn to call to full-time practice those men who began as part-time doctors in small plants; or who have grown up in big ones, as assistant to the chief.

The present graduate might well take stock of himself before deciding to become an industrial physician, asking himself whether he admires big business, is interested in what makes the wheels go round, is truly anxious to better conditions, has a pioneering spirit, is willing to brave misunderstanding—for the job of industrial physician isn't exactly the soft job, the bed of roses so many private practitioners like to believe it is.

A public school education, a good medical college, the summers spent in mixing with working men at their own jobs, preferably in the very industry the young man plans to make his life work, will point the way unmistakably. If, as so often happens, the boy becomes a doctor, is already out in the world before he decides to become an industrial physician, he can take heart. Every bit of experience that he has had will be of use to him. He need not regret a lost year, the year was not lost, but gained, for it has given him a fuller understanding of human nature, which will, in later life, stand him in good stead.

If it is possible that the young man decide what he wants to do while he is still in medical school, he can choose his courses so that they will prepare him for his work with a minimum of confusion.

Once he begins his internship, the cases of occupational injuries which he will see in the clinic, will quickly become of paramount interest to him, and help him shape his own destiny. Once through with school, he should choose an assistantship, or go into private practice helping out with expenses, by doing part-time work for industry. Under no circumstances should he feel qualified to accept a full-time position unless he is working under some man who knows the work well, for it is a disheartening as well as a difficult thing for even an old timer to run a big medical department; for a novice it may well be plain suicide.

Not every man who is well qualified to be a first-rate medical man, can be a first-rate industrial physician. Perhaps a man's attitude towards the job is even more important than the marks he got in medical school, for a good industrial physician is liason officer between employee and employer, and his behaviour, his handling of cases often seems to be more in the department of industrial relations, than it is to iodine and bandages.

Men who have failed in private practice, and who are discouraged and unsure of themselves, often think of industrial medicine as an easy job. Not so! It is one thing to grasp a bull by the horns, but mighty hard to hold on to him. A job in industrial medicine is much the same. If you're not properly qualified, you'd better look out, it may throw you. Private practice has its hardships and its uncertainties, its long hours, its captious neurotics, but industrial medicine has its own problems. Not that I'm going to enumerate them to you, for you are all experts and know all the answers.

No young man should take a full-time industrial job unless he is sure he would prefer it anyway, for the opportunities for part-time work, which will permit of private practice, thus allowing him to taste the bitter and the sweet of each job, before he actually decides on his life-work, are obvious. Indeed it is one way to get a start in private practice, and the more and the better the men are who dip into industrial medicine, the greater will be the gain for industrial medicine.

In every profession where learning is stressed, there are men who seek to get by with a mere smattering of it, and complain bitterly when they are passed by. Actually, about the only young doctor who cannot possibly make a go of industrial work is the know-it-all type who believes that working in a factory for the great unwashed is beneath him. If he is tempted to high-hat the men, he will be shunned by the very men he looks down upon, and lose his job because he is neither competent enough, nor agreeable enough to hold it.

Doctors who are purely men of science, who have no interest in their fellow men, or who are definitely pedantic, are never happy as industrial physicians; and even if they have good jobs, soon leave them to pursue their pet topics, such as intelligence tests, toxicology or major surgery.

Industrial medicine, like any other specialty, means continuous application to study, for never will the physician in industry learn all there is to know about his particular subject. He cannot settle back in an arm chair and dream, burn up the road all Saturday and play golf all day Sunday. He can't get rusty in peace and comfort. He's got to keep up with the times in medical knowledge, a month away from his magazines, and he's got to bone nights like a C- student, to catch up with what's going on in medicine.

He'll have a moderate income to be sure, when it is time to retire, but unless he gives satisfaction to both men and manager, he'll be retired without pay, long before the retiring age. An industrial job is neither a sinecure nor a bid for an old age pension, it is, rather, one of the most interesting and alive specialities that a man of broad interests can take up. It holds surprises, rewards and rebuffs. The best that a good man can do is

sometimes not good enough. Men fail, with the best intentions; but by and large, it is a specialty where merit wins the approbation of both the man at the bench, and the man behind the desk.

In perhaps no other line of work will a doctor come to know men of every class so well, have so broad a contact with his own profession, and, believe it or not, be so free to speak his own mind, and be listened to while he does it!

In conclusion, may I say that with industry putting on a speed-up program, with the draft in prospect, medical men who are interested in men, as well as medicine, will be in great demand. Industry is going to need more and more doctors who can help men turn out more work, who can help men work longer hours without physical failure, for despite the problem of unemployment, there will not be enough trained men for special jobs, not at first, anyway.

Finally, the successful industrial physician can look forward to the same regard from his patients that he would have in private practice. After all, this is the chief reward of any physician.

Diagnostic Factors

— *In Circulatory Disease* —

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IN THE routine or special examination of railroad employees, disease of the circulation is by far the most common malady uncovered. In this two causative factors seem of dominant importance. The first would seem largely to be emotional, including such qualities as a strong sense of duty, responsibility, promptness and alertness. These, perforce, have a stimulating effect upon the vasomotor apparatus, undoubtedly tend to raise blood pressure and increase the wear and tear of the circulation. The second is the age of the employee. Under the rules of seniority, the most responsible positions are held by those in or past middle life and therefore in the age group most subject to vascular disease.

The practical problem of the examiner of the railroad employee is one of prognosis. The questions that must be faced are:

1. How seriously is the circulation of an individual employee damaged?
2. What is the life expectancy?
3. What kind of circulatory accident is to be anticipated?
4. When is it likely to occur?
5. Should fast or slow service be allowed or should retirement be advised?

In seeking solution of these problems the examiner must consider the interests of the travelling public, of the employee, and of the company; interests not always easy to reconcile. It may be said, however, that with knowledge, experience, skill, equipment, and the tact which implies sympathetic understanding of the personal as well as economic factors involved a satisfactory solution usually can be reached. Such a solution demands much of prognosis. It calls for definite statement and precise action based upon prediction of future happenings. Here is prognosis in its most exacting role. Here, then, let us focus attention. In this work we are concerned with two classes of disease of the circulation;

1. Those of infectious origin.
2. Those of degenerative origin.

Foremost in the first class is rheumatic carditis; next in importance is luetic vascular disease. Scarlet fever, diphtheria, and miscellaneous infections rarely are concerned. One has to take into account tuberculosis and the newly recognized vascular disorders presumably of infectious origin—periarteritis, lupus disseminatus, etc.

Let us first consider prognosis in these vascular diseases of infectious origin. In that of rheumatic heart disease one has to consider the age of the patient, the character and degree of the lesion, the size of the heart and its functional response; the evidence of past or present circulatory failure together with its response to treatment.

As rheumatic fever is a disease of early life and as candidates for railroad employment are carefully examined beforehand, very few examples of rheumatic heart disease are found in railroad service. Slight lesions may have escaped the first examiner or lesions may be the result of rheumatic fever occurring after beginning railroad employment. As, of necessity, such occur in adults, cardiac damage of an extensive nature is not likely.

One of the most important elements in the prognosis of rheumatic heart disease is the size of the heart. Since the amount of work required to empty a heart chamber varies with the square of its diameter, the larger the heart the more serious the prognosis. In general, enlargement of the left ventricle is less serious than that of the right. Cases with dilatation of the auricle as well as the ventricle are usually advanced and show signs of failure with a correspondingly serious outlook. The extent and character of the lesions have weight in prognosis. A marked mitral stenosis evidenced by a prolonged diastolic-presystolic murmur at the apex with a snapping, abrupt first sound, a dilatation of the conus and a loud second sound at the pulmonary area with or without hypertrophy of the right heart is not likely to be compatible with active railroad service. The tendency of mitral stenosis to cause such sudden and serious accidents as cerebral or pulmonary embolization should be kept in mind. In railroad work it is not wise to entrust such cases with individual responsibility for others' safety. The same may be said of lesions of the aortic valve, whether they be stenosis or insufficiency and when of marked degree. An aortic leakage accompanied by a Corrigan pulse, a high pulse pressure and great enlargement of the left ventricle rarely is a good railroad risk. One may safely make the same statement about examples of aortic stenosis when there is a large left heart, slow pulse rate, a rough systolic thrill and murmur over the aortic area and a low pulse pressure. In either lesion sudden

Presented at the Seventeenth Annual Meeting of the ASSOCIATION OF SURGEONS OF THE NEW YORK CENTRAL SYSTEM, New York, September 24, 1940.

death from left ventricular failure may occur without the prophetic warning of stasis. When diagnosible, tricuspid lesions mark serious and advanced heart disease and are incompatible with railroad duty. Congenital lesions of the heart play no part in railroad work. Pericardial lesions are likewise so rare in the adult as to be negligible. Rheumatic pericarditis almost never occurs in the adult male. Tuberculous pericarditis occurs more frequently than the rheumatic variety in the male of middle or advanced age.

The value in prognosis of the functional response of the heart has had emphasis from certain cardiologists. While by no means the sole or perhaps the most dependable factor this should be tested in every examination in which the capacity of the circulation is in question. For the examiner in industrial medical work tests of the functional response of the circulation should be neither elaborate nor time consuming. Such tests should not be rigid. Rather should they be adaptable to the physical status of a given patient. I have found a simple exercise test as helpful as any. In this the patient relaxes in a recumbent position for two minutes when the pulse rate and the systolic and diastolic blood pressures are recorded. Then any simple exercise is taken *to the point of beginning dyspnea.* This has seemed more satisfactory than the performance of a measured amount of work. A rough judgment of the amount of work required to bring about slight dyspnea is helpful. The pulse rate and blood pressure are recorded at the end of the exercise and two minutes later after the subject has again relaxed. In a normal functional response, the blood pressure should not rise more than 10 to 15 mm. systolic while the diastolic should remain about the same or should fall some five mm. The pulse acceleration should not last more than two minutes. Dyspnea should disappear promptly. If the pulse remains accelerated, if dyspnea does not subside promptly, and if the blood pressure reacts unduly to exercise, there is a strong argument against the capacity of the circulation to respond to effort. Any indication of past or present congestive failure and especially the response of this to treatment must have weight in the prognosis. Among the minor prognostic aids in rheumatic heart disease are certain instruments — the fluoroscope; the electrocardiogram, and the sphygmomanometer. The roentgen ray serves to check the results of the physical examination in judging of the size, shape and action of the heart. While of value in acute rheumatic disease in estimating the extent and degree of carditis, the electrocardiogram helps little in the prognosis of the chronic phases of the malady. This instrument is of most value here in detecting fibrillation in the cases in which this disturbance of rhythm is in doubt. At this point a word may be said about the general prognostic import of fibrillation in chronic rheumatic disease of the heart. As a rule this disturbance of mechanism attends seriously lesioned hearts and should be a cause of rejection of the employee for responsible service. In exceptional cases, usually with slow pulse rate, no deficit and good myocardial reserve, fibrillation is not incompatible with many years of efficient living, and the employee may be dealt with accordingly.

Syphilitic heart disease is perhaps the most important variety of cases of infectious origin with which the cardiologist has to deal in railroad work. Leutic aortitis with or without aortic insufficiency and with or without aortic aneurysm is the common lesion. Here, again, most of the general factors discussed in the prognosis of rheumatic heart disease come into play. The kind and extent of the lesion, its duration, its rate of progression, the size of the heart chamber, the functional response, the presence or absence of signs of failure, and the response to treatment of such failure or of the underlying disease, all have weight. In luetic cases, however, the emphasis upon the individual factors must differ. Failure of the heart in the luetic cases is likely to be abrupt, is rarely accompanied by fibrillation, ordinarily does not respond well to treatment, and results in sudden or early death. This liability to sudden death of patients with luetic vascular lesions should always be kept in mind by the industrial medical worker. An aortic insufficiency with characteristic physical signs with the background of lues and especially if circulatory symptoms annoy gives a bad prognosis and usually warrants retirement. Cases of luetic aortitis with attacks of nocturnal dyspnea or of anginal pain have a life expectancy of but a few months. The prognosis of aortic aneurysm may be difficult. I have known a case of sacculated aneurysm of the aorta to lead an active life of over 20 years. In general, however, aortic aneurysm is prone to sudden accidents such as are seen in aortitis and aortic leakage. If thus complicated, the need of retirement is suggested. The medical treatment of aortitis and its complications is most unsatisfactory and very little improvement can be expected. Some of the rarer forms of vascular syphilis might be mentioned here. Cerebral hemorrhage in the young without hypertension is quite likely to be due to this disease. The role of meningo-vascular syphilis, or tabes, or of paresis in railroad employees is of no small importance. It is the exceptional case that should not be retired from service. Regional luetic vascular lesions such as aneurysm in other parts of the body may occur but require individual consideration.

Since cardiac damage from such infections as diphtheria and scarlet fever occurs in juveniles, they are not likely to be factors in railroad work. The recent appearance, perhaps more truthfully, the recent recognition of periarteritis, nodosum and lupus erythematosis needs passing mention only. The accompanying symptoms are so severe as to lead to disability even without the benefit of exact diagnosis in their early stages.

The Degenerative Vascular Disease

THIS is arteriosclerotic in nature. The process may be generalized or selectively local in distribution. It may be preceded or accompanied by hypertension or may run its course with normal or low blood pressure. Some of the more important

clinical states caused by arteriosclerosis may be discussed. Among these, disease of the coronary arteries may be given the first place. Common in railroad employees, particularly the enginemen, no other disease is so difficult prognostically or gives the examining physician more moments of indecision. In a few cases this disease may be preceded or accompanied by no functional or organic changes in the cardiovascular tree detectable by any method or methods of examination now at our command. In such the first attack may be fatal. While rare instances of death from coronary disease are seen in the third decade, more occur in the fourth and fifth, while the greatest age incidence is in the early part of the sixth, the peak being at 54. This is an age when the average railroad employee is carrying his greatest load of responsibility.

In view of the liability to sudden death of patients with disease of the coronary arteries, these should not be in railroad service. Practically, diagnosis is more of a problem than prognosis. This is not always an easy matter. The most important factor is the patient's description of his symptoms. Unfortunately the average railroad employee is not a trained observer of symptoms and description may be misleading. An experienced clinician, however, is generally able to elicit a story of cardiac pain in some of its varied manifestations when coronary disease is present. From a prognostic point of view, it may be said that the more frequent the attacks of cardiac pain, the less serious is the condition likely to be. Such a statement seems contradictory but is in accord with clinical experience. A patient with oft-repeated episodes of precordial pressure or pain, with an easily recognized threshold, and with prompt relief from rest or nitroglycerine may have these symptoms many years before graver events result.

It is interesting that cases with hypertension are less likely to suffer the more serious types of coronary disease than are those with normal or low blood pressures. This is especially true of women. Here an element of angiospasm may outweigh that of organic arterial damage.

The physical signs of coronary arterial disease are meager. The heart may be of normal size or even smaller than normal. On the other hand every gradation of enlargement may be found. Ordinarily no valvular lesion is present. One must remember, however, that aortic lesions both rheumatic and syphilitic are likely to be accompanied by anginal pain probably the result of myocardial anoxemia, the result of a lack of adequate filling of the coronary vessels in diastole. While the heart sounds may be normal, they are usually distant, valvular and lack muscular quality. This may in part be due to the rigid chest wall and pulmonary emphysema which so often accompany degenerative vascular disease; in part to degenerative changes in the myocardium. A gallop rhythm in association with heart pain is ominous, as is any evidence of congestive failure. Pericardial frictions point to a recent thrombosis.

Among the accessory instrumental methods of diagnosis the fluoroscope may or may not have value. It may confirm a suspected enlargement of the heart. The shape of the heart as revealed fluoroscopically may be of some prognostic importance especially if it reveals aneurysm of the ventricle or unusual and selective enlargement of any chamber. Evidence of congestive failure is likely to accompany serious lesions of this kind and the prognosis to be correspondingly bad. Elongation, widening or increased tortuosity of the shadow of the aortic arch indicates sclerotic changes which are presumptive evidence of like changes in the coronary vessels. In the diagnosis of disease of the coronary arteries the electrocardiogram plays a great and often a decisive part. When positive electrocardiographic evidence of myocardial damage or of coronary thrombosis is found and especially if serial electrocardiograms show that this is progressive and if these findings fit in with the symptoms and physical signs, the picture is likely to be clear.

In this work one must consider the possibility of a tobacco angina. Although rare, this is a real condition. Ordinarily one finds it in heavy smokers. The anginal symptoms are somewhat atypical. The pain is not always associated with exertion. It may not enforce quiet or be relieved by omitting all effort. It is usually more variable and less consistent than pain of uncomplicated coronary sclerosis. Distress is relieved quite promptly by omitting the use of tobacco and recurs when the use of tobacco is resumed. My own experience would lead to the belief that uncomplicated tobacco angina is rare. In my judgment we should expect an underlying coronary sclerosis the pain threshold of which is lowered by the use of tobacco. In such cases removal of the additional load which tobacco places upon the coronary circulation is enough to abolish the anginal pain.

In general it may be said that the average railroad employee is a well adjusted individual, sound in mind, not prone to anxiety about himself, and highly insensitive to pain. Among these men, therefore, a more or less characteristic story of angina pectoris or of attacks suggesting coronary thrombosis is highly significant. If, in addition, electrocardiographic evidence of myocardial damage with or without supplementary physical and roentgenological findings, is disclosed, the employee should not be entrusted with undivided responsibility for the safety of others.

Hypertensive Cardiovascular Disease

THIS malady might well be called the curse of the railroad employee. It is the chief cause of his disability. In this disease we recognize a dominant inheritance factor, often a temperament marked by enthusiasm, interest, a deep sense of responsibility, anxiety, seriousness, earnestness, and excitability. The first discoverable feature of the disease is an exaggerated response on the part of the blood pressure to stimuli from without or within. At first emergency or in the strict sense, the occasional hypertension gradually becomes continuous. With this are certain organic changes in the cardiovascular tree; a thickening of the

medial coat of the arterioles with hyaline changes and tissue replacement. Thickening of the intima may follow. Tortuosity, narrowing of the arterial lumina and increased fragility follow. These changes at first are dominant in the arterioles; later, the larger arteries may share in the process. The distribution of the arterial changes secondary to hypertension is somewhat capricious. Such may be confined partially or largely to the cerebral vessels, to those of the kidney, heart, mesentery, pancreas, or other parenchymous organs, or to those of the extremities. The initial symptoms are those of vasomotor instability such as tachycardia, flushing, headache, and a mental state characterized by pressure of activity with elements of excitement and fear. As the disease advances, symptoms arise as the result of the structural alterations in the cardiovascular apparatus. These vary with the site, the character and the extent of such changes. On these changes the prognosis as well as the symptomatology depend. To properly evaluate such, the clinician must be prepared to survey the functional and structural variations from the normal of such parts of the cardiovascular tree as are open to examination by any or all methods.

Certain limited parts of the circulatory apparatus are available for this direct or indirect examination and as a result of such examination, the experienced observer, with some degree of accuracy, can evaluate the status of the circulation as a whole. Fundamental must be the recognition that changes in the circulatory tree in vascular disease are selective. It is exceptional to find a case in which the sclerotic or other changes are uniformly distributed. Perhaps the most valuable instrument in this study is the ophthalmoscope. No one can successfully practice internal medicine without being more or less expert in the use of this instrument. Arterial spasticity, changes in arterial light reflex, variations in arterial calibre and contour; constriction, displacement or obliteration of veins at the arterial crossings; patches of degeneration, exudate, hemorrhage or edema; any or all of these are to be looked for. The older patients are more prone to show changes in light reflex and in calibre and contour of the arteries often with "cotton wool" patches, while the younger examples of cardiovascular disease tend to show vessel spasticity and in the later stages extensive hemorrhages, edema and exudate. Ordinarily hemorrhage or exudate suggest a bad prognosis, as do advanced vessel changes. The diminutive cotton wool patches are not incompatible with long life, whereas the large patches of degeneration are of more serious import. If one considers the retina as a prolongation of the brain, it is logical to assume that changes in the circulation here reflect those presumably taking place further back in the encephalon. From advanced vascular retinal changes, therefore, one may assume with reasonable assurance the presence of cerebral arteriosclerosis. The confirmatory evidence of this must be had from study of the patient and of his mental status. Retardation, loss of memory for recent events, errors in judgment, capriciousness, irritability, inability to face new situations, tendency to depression, insomnia; these are characteristic of cerebral arteriosclerosis. When such are present, the changes elsewhere resulting from arteriosclerosis usually are advanced and conclusive. The patient may or may not reveal evidence of cardiac damage in hypertensive cardiovascular disease. In many cases there is a concentric type of hypertrophy without obvious enlargement on either physical or roentgenologic examination. Usually, however, there is an exaggerated apical impulse which tends to be displaced toward the left with an accentuated second sound at the apex and at the aortic area. Tachycardia is usual in typical vascular disease with hypertension. Anginal pain is not very frequent in this vascular disorder. Often it is less serious than anginal pain in a patient with normal blood pressure. The hypertensive subject is prone to angio-spasm which may be a factor in cardiac pain and carries with it a better prognosis than in cases in which structural defects in the coronary vessels are established.

Great enlargement of the left heart, evidence of mitral or aortic insufficiency, which in hypertension usually indicates a dilated mitral or aortic ring, attacks of pulmonary edema which are apt to be nocturnal and which are often called "cardiac asthma" all suggest a bad prognosis.

Certain tests of circulatory efficiency may be of value. The cold pressor test is perhaps a little too elaborate for the routine use of the average busy railroad surgeon. In most cases the simple exercise test already described will serve. In the early phases of vascular disease with hypertension, the effect on blood pressure of exercise to the point of beginning dyspnea is an unusual rise of systolic pressure often as much as 20 to 40 mm. of mercury and a rise of diastolic pressure of 5 to 10 mm. This rise is likely to be very transitory, the pressures returning to approximately the previous figures within two minutes. In the more advanced stages of the disease the return to previous readings is not so prompt, the pressures remaining high for more than two minutes. In the most advanced stages of this vascular disease exercise brings about no changes in the blood pressures. Apparently the circulation has reached the limit of its capacity to accommodate to effort and even at rest the maximum of vaso-constriction is operative. No reserve remains. In cases of this kind the secondary sclerotic changes in the vascular tree are extreme. Almost without exception these include cardiac enlargement with impending or established failure. In a sense the kidney is an important part of the circulation; its structure is largely vascular and the reciprocal relationship of kidney and vascular tree is intimate. The effect of renal lesions, both clinical and experimental, in inducing hypertension needs not be discussed. The results of hypertensive vascular disease upon the kidney may be profound but they are variable. In many instances of essen-

tial hypertension there may be no detectable alterations in renal function and at necropsy only slight hyaline changes in the renal arterioles. The principle of selective or regional involvement of the arteries in hyperpiesis finds expression here. In general, it may be said that the more chronic types of hyperpiesis are less prone to show damage in the renal vessels. It may be noted that such victims are most often found in the older age groups in which death from cardiac failure or cerebral hemorrhage is the rule. Vascular disease in the young may assume the malignant form with arteriolar nephro-sclerosis and death from uremia. Cases of this kind have a very short life expectancy.

The findings upon examination of the urine in the hypertensive may be negative. On the other hand albuminuria may be present in amount roughly proportional to the seriousness of the changes in the renal vessels. Red cells are quite constantly present. In malignant cases recurrent or continuous loss of blood in the urine may be observed. Hyaline casts and in more advanced cases other varieties of casts may be looked for. Retention of uric acid, urea and other nitrogenous products is a late and ominous feature and suggests impending renal or circulatory failure—one or both.

In the prognosis of hypertension, the examination of the available large peripheral vessels is important but rarely decisive. If there is intermittent claudication with diminished or absent pulsation in the arteries of the lower extremities, grave vascular damage is indicated. Mere thickening of the large peripheral arteries such as the brachials, radials and femorals, is not in itself of grave moment. The import of this vascular change must be weighed in conjunction with the other manifestations of the disease.

Such, then, are the factors I have found important in the prognosis of cardiac and of cardiovascular disease as applied to the afflicted railroad employee. To elicit the necessary facts demands accurate observation; to evaluate them calls for discriminative judgment and sound reasoning; to base a decision and recommendation upon them requires that courage of one's convictions which is born of wide experience. Thus qualified the railroad physician may deal with this problem with satisfaction to himself, to the company, to the public and, in at least some instances, to the employee.

History of Fractures

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AN AGE of industrialism and rapid transportation has increased the incidence of fractures a thousand-fold since the days of the hand-weaver and the ox-cart. It is not surprising however, that those who practiced healing in the world's early history should have been able to diagnose fractures and treat them with considerable success. Without the aid of roentgenology, as we enjoy this century, no doubt many of the ancients handled fractures as well as we could do today, if not better. They were compelled to depend upon their natural senses, and thus had developed such aids to the highest degree, whereas we latter day surgeons study fractures almost entirely through the medium of the x-ray. The anatomy of the skeleton was well known, and simple examination usually could recognize the common fractures, especially of the shaft. Thus we read that "a thousand years before Joseph became governor under Pharaoh the Egyptians were using stiffened linen for splinting. It is probable that embalmer's bandage was linen, and gum or plaster was the stiffening agent." No doubt such a method of immobilization was effective, when we witness the stiffness of the materials used in preserving mummies.

The Father of Medicine, Hippocrates, 400 B.C., wrote no better on any subject than on fractures. Many principles, still employed by modern surgeons, were first enunciated by this immortal. His book contains material forgotten by the world for centuries, to be re-discovered in some instances only in comparatively recent times. With few exceptions methods of treatment attained a surprisingly high level, recognition of significant symptoms was remarkably acute, and even prognosis was easily comparable to modern teaching. A carefully prescribed method of traction was used for reduction of fractures of the long bones. This traction was maintained during the application of firm bandages of splints, until the part was adequately supported. For example, in describing the treatment of fracture of the femur, he wrote: "When the thigh bone is broken, particular pains should be taken with regard to the extension, that it may not be insufficient, for when excessive no great harm results from it. For if one should bandage a limb while the extremities of the bone (fragments) are separated to a distance from one another by the force of the extension, the bandaging will not keep them separate, and so the bones will come together again as soon as the persons stretching let go their hold; for the fleshy parts, being thick and strong, are more powerful than the bandaging. The parts should be properly distended (stretched) and put in a straight line; for it is a great disgrace and an injury to exhibit a shortened thigh! For the arm, when shortened, might be concealed, and the mistake would not be great but a shortened thigh-bone would exhibit the man maimed. When then proper extension has been made, you must adjust the parts with the palms of the hands, and bandage the limb in the manner formerly described. The thigh-bone is consolidated in fifty days."

Bandages, splints and fracture troughs were described and their several uses indicated. The nature of the bandage and its method of application were such that it afforded satisfactory support to the fractured limb in a fashion similar to modern plasters. Mixtures of gelatinous substances, clays

Presented at the Third Annual Meeting of the GEORGIA ASSOCIATION OF INDUSTRIAL SURGEONS, Atlanta, September 25, 1940.

and starches were sometimes used to reinforce the bandages. Splints when used were applied with the greatest attention to detail. Speaking further about the treatment of fractured femur Hippocrates said: "A few turns of the bandage should be brought round by the hips and loins, so that the groin and the articulation near the perineum may be included in the bandage; and moreover it is expedient that the extremities of the splints should not do mischief by being placed on parts not covered with bandages. The splints in fact should be carefully kept off the naked parts at both ends; and the arrangement of them to be so managed as that they may not be placed on the natural protuberances of the bone at the knee joint, nor on the tendon which is situated there." Much importance was attached to the length of the posterior support, whether in the form of a splint or fracture trough, since the necessity for immobilization of both adjacent joints in any fracture of the long bones was recognized.

Many joints have been left permanently stiff by too long immobilization, both in this century and in centuries gone by, but Galen (130-200 A.D.) and other surgeons showed modern conception of the value of motion when he wrote: "It should be kept in mind that exercise strengthens and inactivity wastes." He said that an injured limb should not be kept long without motion, but as soon as the inflammation subsides it should be cautiously moved after the application of heat. While mysterious internal diseases may have been treated by invoking witchcraft or doing outlandish things to ward off evil spirits, or by forcing unfortunate patients to swallow horrible concoctions made from the juices of rats and snails and puppy-dog tails, it would seem that the old timers employed common sense in treating fractures. That "exercise strengthens and inactivity wastes" is the simple fundamental statement of all of our scientific knowledge of the physiology of the atrophy of disease. Early mobilization of fractures is one of the many principles recognized by the ancients, ignored for centuries, and in the past 200 years almost perennially rediscovered. One of the errors in treatment of which the ancients were guilty was the idea that fractures should not be fixed at once but should be allowed to wait until the swelling about the fracture had disappeared. Such an idea pervaded the minds of many of the profession even until the present Century, and may be heard today expressed by old-time laymen who ask: "Don't you think you ought to wait, doctor, until the swelling has disappeared before you attempt to set the fracture?"

Through the Hippocratic school many types of fracture-dislocation of the elbow were recognized. In this joint the ease and speed with which contractions occur was well known and early careful motion was strenuously advocated. Regarding fractures of the forearm equal care was taken to prevent malposition. The member was put up at right angles to the elbow, midway between pronation and supination. Pressure sores were considered the result of incompetent practice, and several paragraphs were devoted to instruction for their prevention. First, splints were well padded; second, too firm pressure over the fracture site was to be avoided. They warned against the appearance of gangrene over the heel-cord when the foot was allowed to rest unsupported. They recognized the relationship between prolonged recumbency and bed-sores in the region of the sacrum, and suggested that frequent changes of the patient's position was necessary to avoid them.

The knowledge which these authors of 2000 years ago possessed concerning compound fractures and their surgical difficulties was remarkable. While they knew nothing of bacteria, sepsis and pus were very real to them. They knew, for example, that a wound over a fracture site probably led to bone, and they assiduously avoided probing during the early days following the injury. They felt that such interference caused irritation and provoked the formation of pus, which was long before the days of "laudable pus." To the Hippocratic school there could be nothing praiseworthy about pus of any color or consistency. Those cases in which bone fragments protruded from the skin were known to be more often fatal. Immediate attempts were made to replace the bone ends, and for this purpose various instruments came into play. Either a simple crowbar was used as a lever, or a blacksmith's tongs were used, as a lion's jaw or bone clamp is employed today. Wine and other applications, which today would be recognized as antiseptics, were used freely. They observed that when a wound over a fracture failed to heal, and purulent material began to flow from it in increasing amounts, small fragments of bone (sequestra) would eventually be extruded. A rasparatory, similar to those used by us, was employed to scrape out necrotic pieces of bone. Compound fractures were given much longer periods of rest than simple fractures, were repeatedly dressed, and carefully protected from outside irritation.

A further practice of unusual interest was their use of compression bandages around the limb to promote vascular stasis in cases of retarded union. Harvey did not discover the circulation of the blood until the 17th century, so these pathfinders of surgery could not visualize this phenomenon. The humeral theory was at that time accepted as an indisputable fact, and the object of the compressions was to retain the effusions at the site of injury. To do this they not only compressed the homolateral thigh in the treatment of a fractured leg, but also the entire unaffected lower limb, in order to force the humors into the required area. However, they were careful to avoid prolonged pressure. The use of venous stasis to stimulate the healing of fractures has, with modification of method based on the accepted physiological principles of succeeding eras, been rediscovered with each rejuvenation of medical research since the time of Hippocrates. Thomas, of splint fame, practiced such stasis, and later Bier so popularized it that today his particular method is known by his name. Verily there is nothing new under the sun. As late as 1931 a new research group proved, for reasons consistent with our own biochemical the-

ories that the passive accumulation of blood at the site of a fracture will accelerate healing. Whether the method be compression of an entire limb proximal to the injury, or the proximal and distal application of tourniquets, or repeated local precussion, the object of the procedure remains the same, and its origin dates back to work on fractures generally ascribed to Hippocrates, although there are those who believe that some of the writings attributed to him might have been written by others and only compiled by him.

The so-called "Dark Ages" continued for the first 1500 years of the Christian era, and denoted a lack of progress, if not retrogression, in all phases of life. Nothing was held back more than medicine. The dogmatic teachings of Galen held sway more than the practical ideas of Hippocrates. Galen taught as much that was wrong as was right. He may be justly blamed for postponing so long the discovery of the cause of infection and suppuration. Because Galen said that a certain amount of pus was laudable, all the world believed it and made no effort to disprove it until the advent of Pasteur and Lister. A few workers made half-hearted attempts to overthrow Galen's damaging teaching, but it was so thoroughly entrenched in medical minds that no one would heed anything contrary to it. It is almost unbelievable that a thing so erroneous could survive among intelligent thinking men for so long a time. However, this was only one of the evidences of the "Dark Ages." There were a few progressive intervals. Paul of Aegina (625-690 A.D.) recognized the significance of injury to vertebrae followed by paralysis below the point of trauma. Hippocrates had referred to this lesion but Paul was not quite so pessimistic about the outcome, since he perceived the possibilities of relief in certain cases. Where a fractured fragment of the posterior crural arch is believed to be impinging upon the spinal cord, he wrote, "having first given warning of the danger, we must if possible, attempt to extract by an incision the compressing bone." This is the first description on record of a laminectomy, whether Paul ever practiced it or not. Fracture of the patella also was described in his book. He advocated apposing the fragments by hand and attempting to maintain their aposition by bandaging the limb in full extension. This simple and obvious measure remained the accepted method of treatment until the development of the operative technic in the 19th century.

The Arabians, with Rhazes, Avicenna, and others, followed next, but added nothing to our knowledge of fractures. Salicete (1210-1277) first gave us the definition of crepitus, but is said to have gotten the word from the Hindus. The introduction of cannon shot at the time of the Battle of Crecy in 1346 brought on more compound fractures. The Renaissance in the 16th century saw the arrival of the barber surgeons who were to treat fractures for 200 years; and Ambroise Pare (1510-1590), who devised a system of traction for fractures of the femur, and stated that a fractured patella always led to a stiff knee-joint. He was the first to differentiate fracture of the neck of the femur as a separate clinical entity. Percival Pott (1714-1788) in 1768 described the fracture of the ankle which bears his name. He sustained one of these fractures before he wrote of it. He also described Pott's disease. John Hunter (1728-1793), one of the greatest surgeons of all time, studied the problem of pseudoarthrosis following fractures of the long bones. He observed the smooth eburnation of the fragments, and the formation of articulating surfaces at the bone ends. He realized that this process, in converting rough spongy or cancellous bone into almost hyaline material, would mitigate against union. Consequently he evolved the rational procedure of surgically removing the eburnated ends of the fragments, and irritating the surfaces which would later be apposed. After the operation the extremity was immobilized as carefully as possible until he was certain that union had occurred. This remained the method of choice until the practice of bone-graft surgery came into vogue in the 20th century.

Altogether it may be said that at the beginning of the 19th century the treatment of fractures was much the same as it had been for the preceding 200 years. Immobilization, traction methods, and the care of compound fractures by surgery had been known since the days of Hippocrates, and the introduction by the Renaissance surgeons of France of pulley traction had been almost the only serious innovation since that time. The modern methods of fracture therapy began with the work of Hugh Owen Thomas, of Liverpool (1843-1891). He came from a family of bone-setters of excellent repute, whose secrets, as was usual among this fraternity, had been handed down from father to son for many generations. His father, however, was wise enough to recognize the handwriting on the wall, and in addition to teaching him the art of bone-setting, insisted upon his taking the formal training and license for the practice of surgery. Thomas believed firmly in the therapeutic efficiency of the principle of rest "enforced, uninterrupted and prolonged." To him, stiffness and atrophy were negligible incidentals which in properly handled cases could be corrected with very little difficulty after the bone fragments were firmly united. Such treatment overlooked the lessons taught by Galen, and certainly was not in accord with the methods of modern surgery. However, the value of his practical contributions to the treatment of fractures cannot be overestimated. He was an excellent brace maker, a true surgeon; and his interest in fractures stimulated a great improvement in their care by the profession at large. He established a workshop in his home where braces and splints constructed of metal were made by his hand to designs which varied for each individual case. Among many others he devised the well known and widely adopted Thomas hip-splint, which acquired so much popularity during the first World War. This splint was intended to form an external skeleton for the limb and to transmit weight bearing directly from the tuberosity of the ischium to the ground, thereby relieving the bones of the extremity from deleterious motion or weight bearing. Thomas, however,

made the splint to effect complete immobilization of the lower extremity. It has been subject to numerous modifications, a common form being that of Hodgen, of Kentucky, who eliminated the anterior part of the ring. Hodgen's splint was one of the first to be swung up upon what is now called the Balkan frame, an idea much extended by Blake and others during the World War, and today very much in vogue. In some form Thomas made most of the fracture splints now in use, including shoulder abduction, spinal braces and head elevation apparatus. In 1873 he described a method for using silver ligatures to hold in place the reduced fragments of a fractured mandible.

Dr. W. C. Daniel, of Savannah, was the first to treat fractures of the thigh by the weight and pulley, generally called "Buck's Extension." Daniel's first case occurred in 1819, and his second in 1824. To obtain continuous extension he folded a silk handkerchief around the patient's ankle and tied the ends across the sole of the foot. A cord and weight were then attached, and passed over a roller at the foot of the bed. Later the roller was replaced by a pulley. Dr. Daniel described his cases in the American Journal of the Medical Sciences, August, 1829. Dr. Milton Antony, of Augusta, was impressed by the method, and reported five cases in the Southern Medical and Surgical Journal, October, 1836. It is said that if Crawford Long had reported his discovery of anesthesia in a nationally known journal prior to the Boston surgeons' publication of Morton's work Long would have been given credit for the discovery. The American Journal of the Medical Sciences is one of the oldest and best known of all national journals, and although Daniel published his method of treatment in 1829, and Buck did not publish it until 1852 Buck has been accorded the honor of being the first to employ this useful treatment. Ransahoff, of Cincinnati, first used ice tongs for producing skeleton traction, to be succeeded by the steel nails of Steinmann, to be driven through the bone. In 1814, Colles, of Dublin, Ireland, described the fracture of the lower end of the radius known by his name.

No material for the treatment of fractures has been as universally used as anhydrous calcium hydrate, or gypsum, better known as plaster-of-Paris. Arabian surgeons, and others, had used this substance for centuries, but in the form of molds, not enmeshed in a bandage. The plaster-of-Paris bandage was introduced in 1852 by Mathijsen, Flemish army surgeon. Even as late as the beginning of the present century the bandage was not always applied already impregnated with the plaster, but the plaster was added to the bandage as the bandage was being applied. To complete the discussion of this, the substance most universally employed in the treatment of fractures, the work of the Viennese surgeon, Bohler, should be mentioned. He introduced many innovations in fracture therapy, such as the technic of local anesthesia by injecting novocain into the fresh hematoma of a recent fracture, a procedure which also defied the tradition of the hypersusceptibility of bone to infection, but which has since gained general acceptance. His best known and most discussed contribution is the skin-tight plaster in which plaster-of-Paris is applied directly to the skin. Such a radical method has not been adopted by the profession generally, although this surgeon obtains satisfactory results with it.

It should be remembered that the cases in which Lister first achieved undying fame with antiseptic surgery were compound fractures. Most of the profession, including his own countrymen, thought he was only trying to advocate carbolic acid as a treatment of infection, and were slow to accept his teaching, but carbolic acid was only an incident in the treatment. He was trying to inaugurate a new principle in surgery. He might have used another antiseptic drug just as well, if one had been available. Other Englishmen contributed to better handling of fractures during the latter part of the nineteenth century and the World War period. None was more prominent than Sir Robert Jones, a nephew of Hugh Owen Thomas, former bonesetter.

Another Georgian besides Long and Daniel failed to receive the distinction to which he was entitled. The late J. H. Downey, of Gainesville, invented and used constantly a fracture table many years before the days of Hawley and others for whom such tables are named. In 1910 the author taught fractures in the Atlanta School of Medicine, and learning of Dr. Downey and his work he was invited to demonstrate it before the class. While this apparatus was crude, having been constructed by its inventor, it contained all the principles later shown by Hawley and others in their more finished products. The author urged Dr. Downey to carry his table to New York to have it completed and publicized, but he failed to accept the advice, and never even wrote a paper on the subject.

There is no doubt but that the most useful accessory ever added to the diagnosis and treatment of broken bones is the x-ray, discovered by Roentgen in 1895. It required two decades before the importance of the discovery was recognized universally and utilized in fracture therapy. The writer recalls that probably the first x-ray machine installed in Atlanta was by the progressive dentist, Dr. Thomas P. Hinman, so that surgeons who wished glass x-ray plates made of their fracture cases called on Dr. Hinman. This was about 10 years after Roentgen's discovery, and it was several years later before machines were placed in hospitals and doctors' offices.

Sporadic cases of the open reduction and fixation of fractures appeared in the literature for many years, but it remained for Sir Arbuthnot Lane, in 1905, to standardize such treatment and put it on a firm basis. The drawback lay in the fact that Lane's brilliant experience stimulated unqualified surgeons to attempt to imitate his work, often with serious results, so that the method fell into disrepute. Today, however, the resort to open reduction is a recognized and necessary method, and in the hands of qualified operators, in properly selected cases, is a successful procedure. Lane fixed fragments with plates and screws. The evolution

of internal fixation led to bone-grafting, largely developed by Fred Albee.

Probably no one has achieved greater fame in the non-operative treatment of the serious fracture of the neck of the femur than Royal Whitman, but his method now seems to be succeeded by nailing the fracture under fluoroscopic control. Such a method was in vogue several years ago, and was abandoned, but today a stage approaching perfection seems to be reached so that the best results are being obtained in the history of the injury.

The newest developments in the treatment of fractures fall more under a record of current events than a historical recital. It is realized that many important matters have been omitted in the present narrative. Much should be written about the treatment of fractures of the spine, physiotherapy, and the instruction of laymen in the first-aid management of fractures. While there will be no finality to progress in medicine, it is well always to bear in mind the warning of Hey Groves: "The more the importance of principle and the relative subordinate character of detail are recognized, the better will be the results of fracture treatment."

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The Ageing Process

—*Statistical Notes on Coronary Stenosis and Thrombosis*—

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WE ARE all familiar with that question asked by the patient or his relative: "Doctor, what is the cause of this heart trouble?" As a rule no reply would be nearer the truth than to say "I do not know." But especially the young physician does not dare utter such simple words. The courageous one might say "no one knows exactly." More frequently this truth is clothed in much confusing verbiage. Thus, one day 12 years ago, after leaving the bedside of a patient who had just had an attack of coronary thrombosis, his wife asked me the fateful question. During the pause, as I gathered my words together, I remembered that she had once said to me that it was a sin to have a disease. My reply was therefore based on this point: coronary thrombosis, I explained, is a manifestation of the ageing process and surely, though it may or may not be a blessing, it is no sin to be old. I left that house feeling very satisfied, for this seemed to be a good way of dealing with the question. But next time, the lady asked other questions: "My husband is only 56 years old and other men of this age are playing tennis and working hard," and the inflection of her speech punctuated the sentence with a question mark. I screened ignorance with the comment: "No two people are alike." On this occasion I left the house in a disturbed, dissatisfied mood. This reply was true enough, yet it was not an explanation but rather something to be explained.

The ageing process became a subject of special interest to me, and today I welcome the opportunity of communicating to you some observations and thoughts about it. The poets, novelists and historians have made their contributions. Both general biological literature as well as medical studies of the human being offer a vast amount of information. One finds two schools of thought on the subject of the ageing process. One looks upon it as physiological, the other as a disease process, and reasonable arguments are advanced by both. The late Professor Warthin wrote a good book on the subject of old age only a few years before he died at the age of 75. He had studied and taught morphological pathology for about 40 years before he wrote the following sentences:

"The senescent process is potent from the very beginning, involution is a biologic entity equally important with evolution . . . its processes are as physiologic as those of growth . . . It is, there, inherent in the cell itself, an intrinsic, inherent, inherited quality of the germ plasm, and no slur or stigma of pathology should be cast upon this process . . . Senescence is due primarily to the gradually weakening energy charge set in action by the moment of fertilization."

The late Professor Tilney wrote an article on ageing of the human brain which was published in 1928 the year in which Warthin's book appeared. Tilney, one of America's great neurologists, represents the other school of thought and wrote as follows:

"No evidence thus far adduced is sufficient to convince us that there is such a thing as a strictly old brain. The brain in aged people may present certain morbid changes, but these are in their turn incident to many pathological assaults upon the tissues sustained during life which, in some individuals more, in others less, are in all alike the consequence of infections, intoxications, or other morbid influences . . . All of this is a strong argument in favor of the theory which holds old age in the brain, as in other organs, to be the result of life's successive and accumulative intoxications," and later . . . "According to this view, old age has a pathological background. It arises from definite conditions which may be combated or corrected."

At present one may only speculate about what the ultimate truth may prove to be, but it does seem that what the physician observes is partly physiological and partly pathological. However, the lines of demarcation between these two processes are as yet not clear. It may be added that this is true of conditions at every age period, for we are all familiar with the difficulties of defining

Presented at the Fifty-First Annual Meeting of the AMERICAN ASSOCIATION OF RAILWAY SURGEONS, Chicago, September 18, 1940.

what is "normal" when dealing with so-called borderline cases. There is a fertile field of research in establishing the limits of "normal" with a narrower margin than can be drawn at the present time.

The "normal" duration of life was described as three score and ten years by the psalmists many centuries ago. Our own prophets of today, the statisticians of whom Dr. Louis I. Dublin is a good representative, state that the ideal limit is 100 years but that an average of 75 years is the highest we may expect. In commenting upon the steady increase in "expectation of life at birth" from about 35 years in 1789 to 60 years in 1930, he points out that this is due mainly to reduction of infant and child mortality, and adds that "progress with the diseases of late maturity and old age has not been of any consequence." Since 1930, sulfapyridin has come upon the scene and this will contribute to extending the average duration of life by reducing mortality due to pneumonia and other infections in both young and old. Malignant new growths however still remain a potent cause of death in the fifth and sixth decades of life. Are these due to some neutralizable cause, or are they due to inherited qualities which determine their growth as manifestations of the ageing process?

This question has not yet been solved. Each tissue of the body ages and its functional capacity becomes impaired. Lymphatic tissues which include the widespread lymph nodes, the tonsils, the thymus and spleen, are the first to show signs of involution. The thymus begins to regress before puberty, the tonsil reaches its maximum size at puberty then gradually grows smaller. Lymph nodes in different parts of the body vary in size not only with ageing but, as they are sensitive to acute disturbances in neighbouring tissues, the study of their size in relation to age has been made almost impossible. The spleen reaches its greatest weight in adolescence, then diminishes in size very slowly until the seventh decade and rather rapidly following this. In the digestive system, the reduction in quantity of saliva and in its content of starch digesting enzyme in old age reflects anatomical and physiological alterations. Achlorhydria becomes more and more prevalent after the age of 40, so that at the age of 60 about 35% of people do not secrete hydrochloric acid after taking food. Diverticulosis occurs rarely in persons under 40 and in about 7% of those over 60; this suggests weakening of muscular and elastic tissue with age. These functional impairments do cause symptoms of varying degree but one may live with them far on into very old age. Such anatomical and physiological changes occur throughout the body and at present it cannot be said where the mechanism of control lies or what it is. Among the more obvious signs of ageing are those observed in the cardiovascular system and in the past it has been too readily assumed by some that impairment in nutrition of tissues determines the process of ageing. Hardening and narrowing of arteries results from arteriosclerosis which is mainly a phenomenon of ageing, although it may be partly due to environmental toxic influences. Such abnormal arteries function poorly, or become completely occluded and thus the blood supply to the tissues is impaired or destroyed. The pathological anatomy of such organs as the brain, kidney and heart, seems to justify this view; but in other organs and tissues, as in the glands of internal secretion, the skeletal system and the skin, the manifestations of ageing do not seem to be directly related to the condition of their arteries. Laymen notice the signs of ageing in greying hair and wrinkling of skin; physicians are attracted by evidences of arteriosclerosis. Thus it has become proverbial "that a man is as old as his arteries," and this later became amended, "as old as his coronary arteries."

BECAUSE of progress made in applying methods developed in the physiological laboratory to the clinical investigation of heart action, abnormalities related to disturbances in coronary circulation became a subject of great interest about two decades ago. It may be said that more progress was made in advancing knowledge in this field during the past 20 years than in the time from 1759 when Heberden, of London, first described angina pectoris until 1913 when Herrick, of Chicago, crystallized the syndrome of coronary thrombosis. As one reads the literature on this subject, it appears that about 10 years elapsed before Herrick's work began to be fully appreciated, then the interest grew so rapidly and widely that the diagnosis of coronary thrombosis came to be made too frequently when the condition did not exist. Dr. Herrick himself has played a large role in stabilizing the movement and in the past decade diagnosis has become more accurate. Furthermore attention has been directed to study of prognosis based on close observation of individual cases for long periods of time and the analysis of larger series by the statistical method which Lancelot Hogben so appropriately named "the arithmetic of human welfare." The main result of these studies has been that coronary artery disease is losing its sting. Whereas it was at first considered a highly fatal and totally disabling condition, it is now becoming plain that a considerable proportion of the cases recover and resume their normal activities or some form of reduced activity. My own experience appears to have been similar to that of others who have already published their statistical data.

I HAVE recently completed the tabulation of 11,253 clinical observations made in 6,720 patients from 1926 to 1938, using the Hollerwith system of punched cards and sorting machines. As all the observations were made by myself there is some constancy in source of error. Of course the observer has changed gradually in these 12 years. On the other hand, repeated studies of the same patient offered opportunities for bringing data up to date. Of these 6,720 patients,

there were 549 who had either angina pectoris alone or acute myocardial infarction, or both. The proportion of sexes was three males to one female, this is similar to what has been recorded by many authors in both the Americas and in Europe. It is interesting to speculate why men are stricken with coronary artery disease so much more frequently than women. Every discussion of this kind seems to lead to the demonstration of our lack of adequate knowledge in the domains of genetics and of constitution. Some slight and perhaps interesting clues that I have met with may deserve your attention. Because grey hair is so obvious a sign of ageing, I became interested in that group of individuals who develop this feature early in life, that is before the age of about 30. In both men and women of this type, I have noticed that the edge of the liver is felt at from two to six cms. below the right subcostal border in the midclavicular line, with rare exceptions and at all ages. Both men and women of this type are very eligible candidates for diabetes, arthritis of the non-infectious type, gout, arteriosclerosis, and vascular thrombosis. It is well known that many individuals who develop white hair in the second decade of life live to a ripe old age. Moreover, I cannot offer any satisfactory statistical data to indicate the relation between this constitutional attribute, which in some instances follows the Mendelian law, and duration of life. Yet one cannot escape noticing the regularity with which such persons develop diabetes, arthritis and vascular thrombosis in the fourth and fifth decades. Another observation which may be related to this is that, of the women who have acute myocardial infarction or angina pectoris, the great majority are diabetics. This does not necessarily establish a direct relationship of cause and effect between diabetes and coronary disease, but rather suggests that both these conditions are manifestation of the ageing process in certain individuals and that inherited constitution is the main determining factor. It should be added here that the great majority of patients in the group of 549 with coronary artery disease did not have white hair at a young age, and that no more emphasis is given to these observations than the lack of a large statistical group merits. In this same category falls the observation that those men and women who have grey hairs on the head early in life, do not have them in the eyebrows until the fifth or sixth decade, although hair on face or chest may become grey in the third and fourth decades. This sign very often permits the physician to tell the patient of 40 or 50, whom he sees for the first time, at what age his hair became grey and at the same time the physician directs his own attention to eliciting an accurate account of symptoms related to diabetes, arthritis and vascular thrombosis.

TABLE I

Age	Angina Pectoris (on effort)	Coronary artery stenosis	Coronary artery occlusion with Infarction	Total
20-29	4			4
30-39	15	7	17	39
40-49	40	19	66	125
50-59	68	30	97	195
60-69	49	10	96	155
70-79	6		24	30
80-90			1	1
	182		301	549

TABLE II

104 Fatal Cases: Time between first attack of Coronary occlusion with myocardial Infarction and death.

Time	0-6 Mos.	1	2	3	Years 4	5	6	7	8	9	10-12
No. of Cases	23	9	10	18	11	9	6	7	6	2	3

TABLE III

197 Survivals: Time between first attack of coronary occlusion and myocardial infarction and last observation

Time	0-6 Mos.	1	2	3	Years 4	5	6	7	8	9	10-12
No. of cases	42	35	26	32	20	12	8	11	6	4	1
Working	8	30	18	22	16	10	4	7	2	1	1
Not Working	15	5	8	12	4	2	4	4	4	3	0

TABLE IV

178 Cases: Age in relation to ability to work

Age	Total (table I)	Working Cases	Working %	Not Working Cases	Not Working %
20-29		0		0	
39-39	17	8	50%	4	25%
40-49	66	48	73%	8	12%
50-59	97	40	41%	15	15%
60-69	96	22	23%	29	30%
70-79	24	1	4%	3	12%
Total		119	67%	59	33%

The age distribution in this series of 549 patients with coronary artery disease is not unlike that to be found in the rich literature on the subject. From Table I it is seen that the great majority of cases occur between 40 and 70, and more than half of the cases are in the group of between 50 and 70.

The more difficult questions to answer when facing the patient or his relatives are those related to prognosis. The patient wants to know when he can get back to work, his family are also interested in this, but usually ask how dangerous the condition is. Table II is a basis for answering the question as to duration of life. Thus, of 104 fatal cases, death occurred in the first six months in about 25%.

The duration of life amongst the remainder varied between one and 12 years. The majority lived between two and seven years. This series is not large enough to warrant further analysis, but it seems that the prognosis for duration of life is better in those who have the first attack between 40 and 60 than in those who have it after the age of 60.

The question as to the patient's ability to resume work was from the first answered in a non-committal fashion by saying that if the patient should recover his strength sufficiently it would be more advisable for him to resume work than to remain idle. This was qualified by adding that the work

should be such as he can carry on without any discomfort. The patients who could afford it were treated with massage during the latter weeks of rest in bed and benefited greatly from this; thus, these patients did not experience marked weakness of the limbs which disturbs those who do not have such physiotherapy.

This may account for the fact that of the 197 patients (Table III) whose were living in 1938, 67% resumed some form of work, and only 33% remained idle, some by choice and others because of disabling symptoms. These were due to either angina pectoris on slight exertion or congestive cardiac insufficiency.

Further analysis of this group to determine the effect of age on the ability to return to work (Table IV) reveals that the younger the patient, the more optimistic one may be about saying that he or she will be able to resume a regular occupation.

At the present time this tabulation is of the greatest interest, for it indicates that the advice to resume work rather than remain idle is justifiable. Our data concerning those patients who died does not include the exact description of the circumstances of death in all cases, but those in which we have this information offer evidence in support of the conclusions drawn by Master and his colleagues, to the effect that there is no direct relationship between physical exertion and the occurrence of coronary occlusion or sudden death in cases of coronary artery disease. This contributes to the elimination of unnecessary hesitancy in advising the return to work within limits of physical comfort. Many patients continue at their jobs and use a tablet of nitroglycerine placed under the tongue to prevent or relieve angina pectoris, which may appear on moderate exertion, such as walking to work. It is interesting that these patients are usually free from pain while at work, provided they do not engage in heavy lifting or hurried exertion.

The age distribution of these 549 patients makes it obvious that their coronary artery disease is at least in a very large measure, if not entirely, a manifestation of the ageing process. Viewed in this light it should not be surprising that so large a proportion of these cases were able to resume their economic independence, after recovery from acute illness; for the age process is a slow and gradual one.

In the cases of coronary circulation there is the unique factor of pain associated with the process, which in other organs, such as many parts of the brain and of the kidneys, is painless. Moreover, this pain is often associated with the subjective sensation of impending doom and initiates a natural anxiety neurosis. It becomes the function of the physician to neutralize this neurosis and no words can be more potent than the assurance that the patient will be able to resume a regular occupation, as a rule his usual one. The knowledge that patients have lived for as long as 20 years after the first attack should encourage both the patient and the doctor to view the future with equanimity.

Medical Service in Industry

THE following article is the Introduction to the series of articles on "Medical Service in Industry" which, prepared with the approval of the Council on Industrial Health, will appear from time to time in the *Journal of the American Medical Association*. This is from the *Journal* for September 28, 1940:

LARGE industrial establishments in this country are classified as those which employ more than 500 workers. Many business organizations of this size have found medical supervision over employees, materials, processes and working conditions to be a practical necessity. This trend gains strength an industrialists learn by education and example that maintenance of trained manpower at gainful employment uninterrupted as far as possible by disability from injury or illness is good business. It is possible to demonstrate, as many physicians have done over long periods of time and in all kinds of manufacturing operations, that industrial medical departments can be conducted without compromise of ethical or scientific medical standards and yet in excellent conformity with the purposes of industry. Once this view is widely accepted by industry and the medical profession, greater demand for the services of physicians as a means for conserving the health of workers may be expected.

DISTRIBUTION OF INDUSTRIAL ESTABLISHMENTS ACCORDING TO NUMBER OF EMPLOYEES

Establishments Employing	Number of Establishments	Number of Wage Earners
Number of wage earners	6,885	.....
1 to 5 wage earners	62,164	170,174
6 to 20 wage earners	46,402	514,487
21 to 50 wage earners	23,138	750,922
51 to 100 wage earners	11,911	852,373
101 to 250 wage earners	9,745	1,522.670
251 to 500 wage earners	3,911	1,363,000
501 to 1,000 wage earners	1,660	1,333,323
1,001 to 2,500 wage earners	737	1,080,534
2,501 or more wage earners	241	1,181,748
Totals	166,794	8,569,231

U. S. Census of Manufacturers, 1937.

These demands will probably come soonest from large firms able individually to support adequate medical facilities, yet which have for various reasons failed to develop them. A more important demand should come from small manufacturing units and other commercial enterprises, once a means is found for providing an adequate service which they can with advantage support. The accompanying table describes the distribution of industrial establishments classified according to the number of wage earners employed.

Frequently there is great need for medical service in these small plants, the more so since it is unrealized and since unfavorable sickness or accident experience is not in any sense a function of plant size. The Council on Industrial Health believes that these needs represent a considerable opportunity for medical achievement and that this demand should be encouraged and controlled through the establishment of an educational service which will:

1. Acquaint physicians with the special character of industrial medicine, surgery and hygiene and the necessity for the enforcement of standards defining scope and professional competence.

2. Convince employer and employee alike that medical service in industry merits support only as it is soundly organized economically, scientifically and ethically and that to produce lasting benefits it must be satisfactory both to those who provide and to those who receive the service.

Manufacturers' associations, trade unions, insurance carriers and compensation and other governmental agencies have large and legitimate interests in the workers' safety and health. All are actively engaged in establishing economic, social and humanitarian values of control over industrial accident and disease. In view of this widespread interest the ability of medical organizations to exert a constructive influence on industrial health standards takes on added significance, since other agencies are not in good positions to evaluate medical procedure or facilitate direct application of improved knowledge or technic into actual practice. To relinquish leadership in any program involving the public health to extraprofessional agencies is out of keeping with medical tradition long established in other specialty fields. Principal reliance for improved industrial health standards must rest on medical education and self discipline administered through the physicians' own organization. The Council on Industrial Health of the American Medical Association was established as a means of focusing attention from all elements in the medical profession on industrial health problems. For identical reasons cooperating communities on industrial health are being organized in the medical societies of states where the nature or concentration of industry appear to justify such a step.

Functions of State Committees on Industrial Health

A PROGRAM has been developed as a guide to committees on industrial health in state medical associations which has the following well defined objectives:

1. To train industry and labor to the value of industrial health conservation.
2. To develop a clear understanding of the proper scope and functions of industrial medicine and to clarify relationships between private and industrial practice.
3. To keep the medical profession informed about all accepted methods for reducing the frequency and severity of industrially induced disability.
4. To elevate medical relations under workmen's compensation.
5. To scrutinize all legislation affecting the health of industrial workers.
6. To improve relationships between medicine and insurance.
7. To establish working relationships with all agencies in the state interested in industrial health.
8. To arrange for the adoption of similar activities through cooperating committees in the medical societies of the industrial counties.

To accomplish these objectives it has been recommended that the personnel of the state committees include representation from:

1. Private practice.
2. Industrial medical practice.
3. Medical represenation if such exists from each of the following:
   (a) State bureau of industrial hygiene.
   (b) State workmen's compensaiton agency.
   (c) The medical faculties in the state.
   (d) Industrial insurance company.

A bulletin has been developed by the Council on Industrial Health to permit interchange of ideas between committees of the state societies and as a means of estimating progress.

County Medical Society Organization

THE adoption of similar objectives and methods of organization by cooperating committees in county medical societies provides a sound basis for mobilizing community resources for general or special industrial health actvity in accordance with local needs. Under such a plan it should be possible to establish sources of information about the character of local industrial operations and the important health problems of each as a first step toward early recognition and control. The resources of other community organizations interested in industrial health need investigation in order to determine how they may contribute to physical betterment of workers under enlightened medical leadership. There will be many opportunities to promote better professional training. The general public can be informed of accomplishments in industrial practice and their effect on the health of the community as a whole. An excellent means is provided whereby the interdependence of private and industrial practice is demonstrated and the activities of both augmented by consulting services in industrial hygiene developed by governmental and private agencies. Working relationships can be promptly established with the allied professional groups—industrial hygienists, safety engineers and industrial nurses.

Recent developments indicate that county society committeee organization of this character can contribute materially to the provision of industrial health services for the small plant owner and his employees interested in deriving benefit from advance in industrial medicine, surgery and hygiene but who in the past have found themselves unable to do so. Most important, it establishes a direct means of assistance to the physician who wants to know how he should conduct himself for greater and more competent participation in this field.

American Conference on Industrial Health

A SYMPOSIUM on the Practical Value and Application of Industrial Health Work, will be held by the AMERICAN CONFERENCE ON INDUSTRIAL HEALTH, at Chicago Towers, 505 N. Michigan Ave., Chicago, on November 14, 1940, with the following tentative program:

Morning Session:

DR. C. D. SELBY, Medical Consultant, General Motors Corporation, Detroit, *Presiding*:

FROM THE VIEWPOINT OF:

The Employer—MR. B. C. HEACOCK, President, Caterpillar Tractor Co., Peoria, Ill.

The Physician—DR. WILLIAM A. SAWYER, Medical Director, Eastman Kodak Company, Rochester, N. Y.

The Industrial Hygienist—MR. WARREN A. COOK, Zurich Insurance Co., Chicago.

The Employer—MR. D. A. MOORE, Western Electric Company, Chicago.

The Insurance Company—MR. AMBROSE KELLY, American Mutual Alliance, Chicago.

The Legal Profession—MR. FRANK PEREGRINE, of Peregrine and Bruegger, Lawyers, Chicago.

Labor—MR. MATTHEW WOLL, Secretary, American Federation of Labor, New York.

Luncheon Session:

DR. C. O. SAPPINGTON, Consulting Industrial Hygienist, Chicago, *Presiding*:

"The American Conference on Industrial Health"—DR. VOLNEY S. CHENEY, Medical Director, Armour and Company, Chicago.

"Industrial Health and the National Defense"—DR. PAUL A. NEAL, Chief, Division of Industrial Hygiene, U. S. Public Health Service, Washington, D. C.

Afternoon Session:

DR. E. C. HOLMBLAD, Medical Consultant, Railway Express Agency, Chicago, *Presiding*:

FROM THE VIEWPOINT OF:

Employer-Employee Relations—MR. W. E. ODOM, W. E. Odom Associates, Chicago.

Finance and Cost Accounting—MR. A. W. TORBET, C.P.A., of Frazer and Torbet, Chicago.

Employer Selection and Rating—MR. EUGENE BENGE, Chicago.

Safety—Mr. F. A. Lauermann, Republic Steel Corporation, Chicago.

Public Relations—Mr. Ralph L. Lee, Department of Public Relations, General Motors Corporation, Detroit.

Nursing—Miss Joanna M. Johnson, Supervisor of Nursing, Employers Mutuals of Wausau, Wisconsin.

Research—Dr. Leroy U. Gardner, Director, Saranac Laboratory, Saranac Lake, N. Y.

Industrial Psychiatry—Dr. Lydia Girerson, Metropolitan Life Insurance Co., New York.

Dinner Session:

Oliver E. Mount, Secretary-Treasurer, American Steel Foundries, Chicago, *Presiding*:

"Industrial Health and the General Practitioner"—Dr. Morris Fishbein, Editor, *Journal of the American Medical Association*, Chicago.

"The Humanitarian Appeal of Industrial Health"—Major John L. Griffith.

The American Conference on Industrial Health was recently incorporated, not for profit, under the laws of the State of Illinois. This, its first meeting, is sponsored by the American Association of Industrial Physicians and Surgeons, in collaboration with other interested groups.

Industrial Health Symposium

A SYMPOSIUM on Industrial Health and Safety will be held in Saginaw, Michigan, November 12, 1940. This is sponsored by the Saginaw County Medical Society, under the supervision of the Industrial Health Commitee. There will be morning sessions devoted to the relations of the manufacturer and industrial health; a luncheon meeting, regarding industrial health and the national defense; afternoon sessions given over to personnel problems in industrial health; and the evening dinner meeting concerned with a primarily medical industrial problem. Dr. R. D. Mudd, Medical Director, Chevrolet - Grey Iron Foundry, is Chairman of the Industrial Health Committee.

Air Hygiene Foundation

INDUSTRIAL Health Defense will keynote the fifth annual meeting of Air Hygiene Foundation to be held at Mellon Institute, Pittsburgh, November 12-13. The impact of stepped-up production on health in the mining, mechanical and chemical industries will be stressed.

Progress reports are scheduled on the industrial health research which the Foundation supports at Harvard, Saranac and Penn. Investigations cover such subjects as health protection in electric welding, individual susceptibility to dusts, and improved measures for control of health hazards.

Other reports will deal with the Foundation's program for reducing sick absenteeism in industry and with current legal trends affecting compensation and occupational disease.

A special symposium is planned on health problems arising in the chemical and mechanical industries bearing the brunt of defense production.

Tick Fevers

John D. Shingle, M.D.,
Cheyenne, Wyoming

ROCKY Mountain Spotted Fever is primarily a disease of small rodents. It is not highly fatal to these animals. It is also transferred from tick to tick by latent infection in the larger wild and domestic mammals. Man is not primarily host for the tick, and it is only an accident of nature when the human contracts spotted fever.

Spotted fever has been known in this country as a clinical entity since 1873 when it was reported in the Snake River Valley. At that time it was called the "Fever of Idaho," and was the subject of a publication in *The Medical Sentinal* in 1899. In 1902 an investigation was made of the disease as it occurred in the Bitter Root Valley of Montana where the mortality rate was between 70 and 80%. It was during this investigation that the tick Dermacentor andersoni was definitely incriminated as the vector.

When spotted fever was first identified it was thought to be geographically limited strictly to the mountainous section of northwestern United States. As late as 1930 it was considered to be definitely limited to the states west of the Mississippi. In 1930 an investigation was carried on by Spencer and Maxcy on the epidemiology of endemic typhus on the eastern seaboard. They found that many cases that had been reported as Brill's disease were not typhus but spotted fever. This immediately extended the known area of spotted fever infection until now cases have been reported from almost every state except those in the New England section. In fact more cases of spotted fever are reported annually from the states of Delaware, Maryland, Pennsylvania, Virginia, North Carolina and the District of Columbia than in all states west of the Mississippi combined.

The Rickettsia of spotted fever are transferred by the bite of the tick. In the Rocky Mountain region it is the wood tick Dermacentor andersoni. On the eastern seaboard it is the dog tick Dermacentor variabilis. These parasites can harbor the infection for years and once infected they are always infectious. Ticks are infected by the transmission of the Rickettsia through the egg to the nymph to the larvae to the imago without intermediate hosts. The nymphal and larval forms feed on the rodents and an infectious individual will spread the Rickettsia to other immature ticks which subsequently feed on the rodent. The ordinary life cycle of the tick is about two years but those which do not secure food can live for a period of five years with suitable shelter.

Man is bitten only by the adult tick which appears in the spring in large numbers and generally disappears by July first. Consequently very few, if any, authentically established cases of spotted fever occur after this date.

The disease is one which occurs in all types from a latent, sub-clinical, unrecognized form to a ful-

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• Presented at the Fifty-First Annual Meeting of the American Association of Railway Surgeons, Chicago, September 17, 1940.

minating, rapidly fatal type. Most cases have a typical symptomatology with the fatality rate varying with the geographical origin of the infection. In extremely mild attacks without rash the diagnosis is presumptive. The majority of these people are never seen by the physician. The ambulatory patient has a low-grade fever with a scanty rash which may be either scattered or localized. The duration of the rash is less than two weeks and the patient is not sufficiently ill to require bed rest. In the abortive attack recovery is complete in from three or four days to one week. The temperature may go as high as 104° and the pulse rate to 130. The clinical symptoms subside rapidly and rash disappears in about two weeks.

The fulminating, malignant infections are so highly toxic that the body defenses are overwhelmed and death occurs in from three to five days. The majority of patients dying from spotted fever succumb in the first week of their illness. In these instances there may be no rash or the spots may coalesce to form large ecchymotic blotches. There is early involvement of the central nervous system with delirium and aphasia.

THE more typical infections are vastly in the majority. The incubation period varies from two to five days in the more severe cases to three to 14 days in the milder ones. A prodromal period of two or three days or longer may occur characterized by anorexia, irritability, malaise and chilly sensations. The onset is usually sudden. The symptoms most often complained of are frontal and occipital headache, intense aching in the lumbar region and marked malaise. These may be accompanied by chill, sweating, injection of the conjunctivae, sensitivity of the eyes to pressure, photophobia, pain in the upper part of the abdomen, pain in the bones and muscles or both, (and this is a usual manifestation), nosebleed, nausea and vomiting. The symptoms increase in severity according to the virulence of the infecting strain. Chilling may continue. The face soon assumes a typical dusky flush often with an apprehensive expression. At times there is a definite body odor. Cyanosis is frequent. The pain in the muscles and bones may diminish with the appearance of the rash. The spleen is enlarged and, if palpable, can often be felt by the third day. Edema if present, may be generalized, but is more frequently limited to the extremities or the face.

The typical rash usually appears from the second to the fourth day but may be delayed 24 to 48 hours. It is sometimes preceded by a mottled appearance of the skin and is most often observed first on the wrists and ankles and less commonly on the forehead spreading in a centripetal fashion. The color varies from pale to bright rose. The larger and more scattered the spots and the brighter their color remains as the disease progresses the better is the prognosis.

The lesions are commonly macules, but a papular form sometimes occurs. The eruption extends rapidly to all or most of the body including the palms of the hands, soles of the feet, the scalp and sometimes even the eyelids and mucous membranes of the mouth and pharynx. The rash in full development is purpuric and as a rule is most abundant and most intense on the wrists and ankles, the legs, the upper part of the back, the lateral surfaces of the arms and the buttocks.

The rash is the visible evidence of the specific lesions in the walls of the blood vessels. These lesions are characterized by the proliferation of the endothelial cells, local necrosis of the endothelium and smooth muscle, and thrombosis. In the more severe attacks and in fatal cases in which death is delayed there may be resultant sloughing, the parts commonly affected being the scrotum, prepuce, vulva, dependent portions and buttocks.

The febrile period is usually from two to three weeks but may be shorter or longer. Its course is much like that of endemic typhus, but it reaches higher levels. The rise in temperature is slower in milder attacks and the maximum may not be greater than 103° F., while in the highly fatal type the temperature rises more rapidly and to a maximum of 104° to 106° F. More or less marked morning remissions are the rule. The maximum temperature usually persists into or through the second week. The fever declines by lysis reaching normal about the middle or end of the third week. In patients infected with variabilis strains, lysis is usually accomplished in three to four days, but with the andersoni strains lysis frequently extends over seven or eight days. In fatal attacks terminal hyperpyrexia is occasionally observed, the temperature reaching as high as 108° F.

The pulse is at first full and strong and the rate in less toxic patients may not exceed 90; with greater toxicity it tends to become shallow and the rate may range from 110 to 160. A high pulse rate and high temperature soon after onset are unfavorable signs. The lungs usually are not involved, but a slight hacking non-productive bronchial cough is typical. Restlessness and insomnia are prominent. Hyperesthesia of the skin and neuritis of varying degrees are common. The mind may remain unusually clear even in fatal attacks but more often there is stupor from which the patient is easily aroused, or low grade or active delirium. Patients who appear to remain clear mentally may later have no recollection of the events of their illness. Ankle clonus, a Babinski sign, and Kernig's sign may be present, and a slight nuchal rigidity is not uncommon.

The tongue is coated, but remains red at the edges and may be swollen. Persistent constipation is usual, but in some cases intestinal attacks probably result from involvement of the capillaries of the intestines. Vomiting is most common during the first few days but may occur later. The liver may be enlarged. Jaundice is not uncommon and sometimes is a marked terminal condition.

The urine is usually reduced in amount—the greater the reduction the poorer the prognosis. It is acid and at times strongly so. Albumen and acetone may be present, especially in the more severe cases. There may be either incontinence or complete retention.

Beyond a white count of 1500 with a relative mononucleosis, the pathologic changes in the blood are not remarkable. The spinal fluid is normal.

In endemic areas where the only prevalent rickettsial infection is spotted fever, clinical diagnosis is remarkably accurate. When spotted fever and endemic typhus co-exist the differentiation is not easy and laboratory methods of diagnosis are not only well advised, but usually essential. The three tests ordinarily used are the infection test, the Weil-Felix reaction and the protection or virus neutralization test. On the whole the Weil-Felix reaction which makes use of the fact that agglutinins produced in the patient's serum by the virus cause clumping of the OX-19 strain of Proteus vulgaris is the most useful and practical.

The complications of the disease which are most frequently encountered are bronchial, lobar or hypostatic pneumonia; phlebitis; hiccough; and hemorrhages from the nose, intestine or kidneys. Iritis, acute glomerulo nephritis and hemiplegis occasionally appear. Convalescence is slow and complete recovery may require from a month to a year or even longer. This is true of even relatively mild infections. Concurrent conditions due directly or indirectly to ticks may occur such as streptoccocic septicemia, tularemia and tick paralysis.

The sequelae following an attack of Rocky Mountain spotted fever include deafness, impaired vision, insomnia, neurosis, anemia, tachycardia, and thrombo-angiitis obliterans; and in the gastro-intestinal form, partial intestinal obstruction. The progress of degenerative diseases in persons of more advanced years may be unfavorably affected. In some instances those who recover from severe infections seem to have less endurance and occasionally the mentality seems less acute.

There is no natural immunity to the disease, and all races are susceptible. Previously it had been considered that one attack of spotted fever provided a life long immunity, but recent data suggests that the degree and duration of the resistance are in more or less direct proportion to the severity of the infection. At any rate, it seems certain that mild involvement does not always give permanent protection.

THE treatment is mainly symptomatic and supportive. Prompt and energetic measures often bring about a successful termination even in cases which appear essentially hopeless when first seen. Among these measures, intravenous glucose appears to have definite value. There is, however no known specific. The use of neosalvarsan dissolved in metaphen and given intravenously has been suggested, but further work is required before its value can be definitely determined.

Complete prevention of spotted fever is accomplished only by the avoidance of tick bites. For persons living in tick infested regions this is impossible. For these areas, data accumulated from 15 years' of experience indicate: That vaccine has a definite immunizing value; that persons infected with a virulent strain have a definite alleviation of symptoms; that infections by mild strains are usually asymptomatic; that immunization seldom exceeds one year but that each subsequent vaccination affords a higher degree of immunization. In areas where relatively mild infections predominate and the incubation period is often more prolonged, the administration of vaccine as soon as possible after tick bite may ameliorate an impending infection. This procedure, however, is not recommended in areas of highly fatal infections where the incubation period is short, unless the patient has been previously vaccinated.

ROCKY Mountain spotted fever is sometimes confused with typhoid, measles, smallpox, post-measels encephalitis, purpura hemorrhagica, endemic cerebrospinal meningitis, secondary syphilis, endemic typhus and Colorado tick fever, the latter causing the most difficulty in the mountain areas.

In these regions, febrile reactions, which do not appear to be the result of rickettsial infections, are of relatively frequent occurrence following the bite of the tick Dermacentor andersoni.

This disease as generally reported is of a remittent type and unlike spotted fever is characterized by two febrile periods, no rash, a general appearance of tenseness, and tenderness of the calf muscles of the leg. The remaining symptoms simulate spotted fever. Colorado tick fever has been reported as a mild type of spotted fever. However, this does not seem likely. The onset is more sudden than in mild cases of spotted fever, the pulse rate accelerates more rapidly and the fastigium is reached more quickly. The initial pains and generalized malaise and backache are usually more intense; also, as stated before, there is no rash. Furthermore, all attempts to reproduce the infection in laboratory animals have consistently failed yet can be easily accomplished with spotted fever; and finally sera from blood samples taken both during illness and convalescence have not agglutinated Proteus OX-19 organisms in significant titer.

IN CONCLUSION may I say that when tick bite is followed by a single short febrile period and there is no rash, no local lesion at the site of the tick bite, and glandular involvement is absent or slight, the diagnosis is often difficult. There are five possibilities: Mild Rocky Mountain spotted fever, tularemia, Colorado tick fever, toxemia, or some other condition of unknown etiology. The likelihood of Rocky Mountain spotted fever is presumably a slight one.

Tularema is a definite possibility, though it is impossible at the present time to state what proportion of the type of cases in question can be thus accounted for. Certain it is however that mild atypical cases of tularemia, without local lesions and a slight or perhaps no glandular involvement do occur following tick bite. Diagnosis in these instances can always be established by agglutination tests. Whether or not one-febrile-period cases of Colorado tick fever occur is felt to be an open question, since it is known that there are cases of apparently purely toxic nature in which the symptoms are similar.

Industrial Medicine
Reg. U. S. Pat. Off.

The Journal of Traumatic Surgery and Occupational Diseases

Established 1932, by A. D. Cloud

Executive and Publication Offices:
413 Pleasant St., Beloit, Wis.

Eastern Offices: 110 East 42nd St., New York City.
Editorial Offices: 540 No. Michigan Ave., Chicago.

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VOLUME 9 OCTOBER, 1940 NUMBER 10

Reciprocal Analysis

COLONEL TUTTLE'S "Personnel in Industry" (page 500) has a sentence which, it seems, pretty well crystallizes the philosophy of one major phase of industrial medicine. This refers to "the importance of selecting the right man for the right job, *based on a reciprocal analysis.*" (Our italics.) And Dr. Woody's "Industrial Medicine as a Specialty" (page 510) has a sentence which, it also seems, very clearly highlights the italics. He says: "Is industrial medicine really a specialty? Is there a need for it? Emphatically we all say: 'Yes,' but as you know, many important and able physicians disagree with us." But why do they disagree? As the Council on Industrial Health says (page 524): "It is possible to demonstrate, as many physicians have done over long periods of time and in all kinds of manufacturing operations, that industrial medical departments can be conducted without compromise of ethical or scientific medical standards and yet in excellent conformity with the purposes of industry." That removes the principal reasons for the general disagreement so long persisted in by many. No one will argue with the advantages of "the right man for the right job." The remaining disagreement, therefore, must be in respect of the "reciprocal analysis" — failure or refusal to comprehend its significance! We mentioned a long time ago in these columns the M.D. who wouldn't accept suggestions as to the importance of examining applicants for a certain employment with respect to the requirements of that employment, saying: "An examination is an examination . . . !"

Two Important Committees

TO THOSE who heard, or read, Dr. Heiser's discussion of Mr. Nelson's "Pre-Employment Examinations" in Industrial Medicine for September, 1940, page 456, the Committee on Pre-Employment Physical Examinations, which has been named by Secretary of Labor Frances Perkins, presents an interesting personnel. It is composed of 16 persons:

Representing Labor: Robert J. Watt, International Representative, A.F.L.; John P. Frey, President Metal Trades Dept., A.F.L.; Herbert W. Payne, Textile Workers Org. Committee; Thos. F. Burns, United Rubber Workers.

Representing Management: W. H. Winans, Union Carbide Co.; V. P. Ahearn, National Sand and Gravel Assoc.; Dr. Ralph Greene, Eastern Airlines; Dr. Harvey Bartle, Pennsylvania Railroad Co.; M. M. Olander, Owens Illinois Glass Co.; Victor G. Heiser, M.D., N.A.M.

Representing the Public: Cyril Ainsworth, American Standards Assoc.; Prof. Philip Drinker, Harvard University; I. S. Falk, Social Security Board; Martin P. Durkin, Dir., Illinois Dept. of Labor; O. F. McShane, Utah Industrial Commission; Dr. Leo Price, International Ladies Garment Workers.

ANOTHER Committee which has a timely importance is the A. A. I. P. & S. Committee "for the development of a Health Program in Industry concerned with National Defense." This was created under the following not undramatic circumstances:

At the opening of the Twenty-Fifth Annual Meeting, at New York, on June 6, 1940, Dr. McIver Woody, President of the Association, said: "I would like to read, just as it was presented to the Board of Directors Monday night, an official request in the form of a telegram from Thomas Parran, Surgeon-General of the United States Public Health Service, in his official capacity: 'In connection with the National Defense program, the Public Health Service desires the American Association of Industrial Physicians and Surgeons to appoint a committee to cooperate with the Division of Industrial Hygiene of the National Institute of Health in developing and applying plans or programs for coordinating the health need in industry.' The Board of Directors authorized the following telegram: 'In compliance with your request, the American Association of Industrial Physicians and Surgeons by unanimous vote of the Board of Directors has authorized me to appoint Dr. Royd R. Sayers, Dr. Clarence D. Selby, Dr. Edward C. Holmblad, Dr. Daniel L. Lynch, Dr. T. Lyle Hazlett, Mr. William P. Yant, and Mr. Philip Drinker as a committee to cooperate with the Division of Industrial Hygiene of the National Institute of Health in plans or programs for coordinating the health need in industry'."

INDUSTRIAL HEALTH AND DISEASE

The Common Cold

—Prophylaxis by the Oral Route—

PAUL FORGRAVE, M.D., and JOHN FORGRAVE, M.D.,
St. Joseph, Missouri.

RECENT surveys made by the U. S. Public Health Service, industrial physicians and insurance companies indicate that an average of more than 50% of the time lost from work is due to colds and their complications. While such time lost is not covered by compensation insurance and is not chargeable to the employer, the employer does suffer the inconvenience and loss of efficiency incurred by having to substitute less skilled workers for those regularly trained to serve their particular function. The treatment outlined below is so inexpensive and so easily administered that employers might find it profitable to furnish it to their employees either free or at cost.

Since 1933 considerable work has been carried out in regard to oral vaccination against the common cold. The value of oral vaccination has been conclusively demonstrated in recent years by controlled experimental administration to groups of people who live and work in the same environment. Most of this work has come from the Eastern and Southern portions of the United States and not from the West or Middle West where many large industrial plants are located. It was for this reason that a small group of workers from a Mid-Western Plant (Quaker Oats Co.) were immunized by the oral route and accurate records kept to see if climatic conditions would have any effect on the end result of this type of vaccination against the common cold.

The industrial plant, where all individuals in the experiment were employed, is concerned with the production of several types of breakfast foods and a well-known pancake flour. The atmosphere in this type of plant is therefore necessarily filled with grain particles which would be quite irritating to the mucous membranes of the upper respiratory passages. It might be concluded that individuals employed here may be more susceptible to infections of the nose and throat than if they were employed elsewhere. This conclusion is given more weight from the fact that time lost due to illness during the Winter of 1938-39 was, in over 50% of the cases, due to the common cold.

The medication used in this work was an improved vaccine prepared in the form of small, white tablets for administration by mouth. It might be well to note that the tablets were enteric coated to prevent destruction of antigens by stomach secretions. This assures their arrival in an unchanged state into the intestine, where absorption takes place. The tablets were prepared from selected bacterial strains of recent isolation. Each tablet* contained a total of 50,000 million killed organisms, as follows:

	million
Pneumococci (D. pneumoniae) Types I, II, and III.	12,500
Micrococcus (Neisseria) catarrhalis	10,000
Influenza bacillus (H. influenzae)	10,000
Streptococci (mixed)	7,500
Friedlander bacillus (K. pneumoniae)	5,000
Staphylococcus aureus	2,500
Staphylococcus albus	2,500

The method of administration was as follows: one tablet 15 to 20 minutes before lunch daily for seven days; two tablets a week until 20 tablets had been taken, then once a week for four more doses. The work was started on October 16, 1939. The time of administration, i.e., before

*Catarrhal Oravax (Merrell)

TABLE I

EFFECT OF ORAL VACCINE ON INCIDENCE OF COLDS

	Experimental Group (Oral Vaccine)	Control Group (No Vaccine)
Number of workers	40	40
Average number of colds in previous seasons	4.85	4.2
Average number of colds during experimental period	1.45	3.9
Decrease in number of colds in experimental period	3.75 (70.2%)	.3 (7.2%)

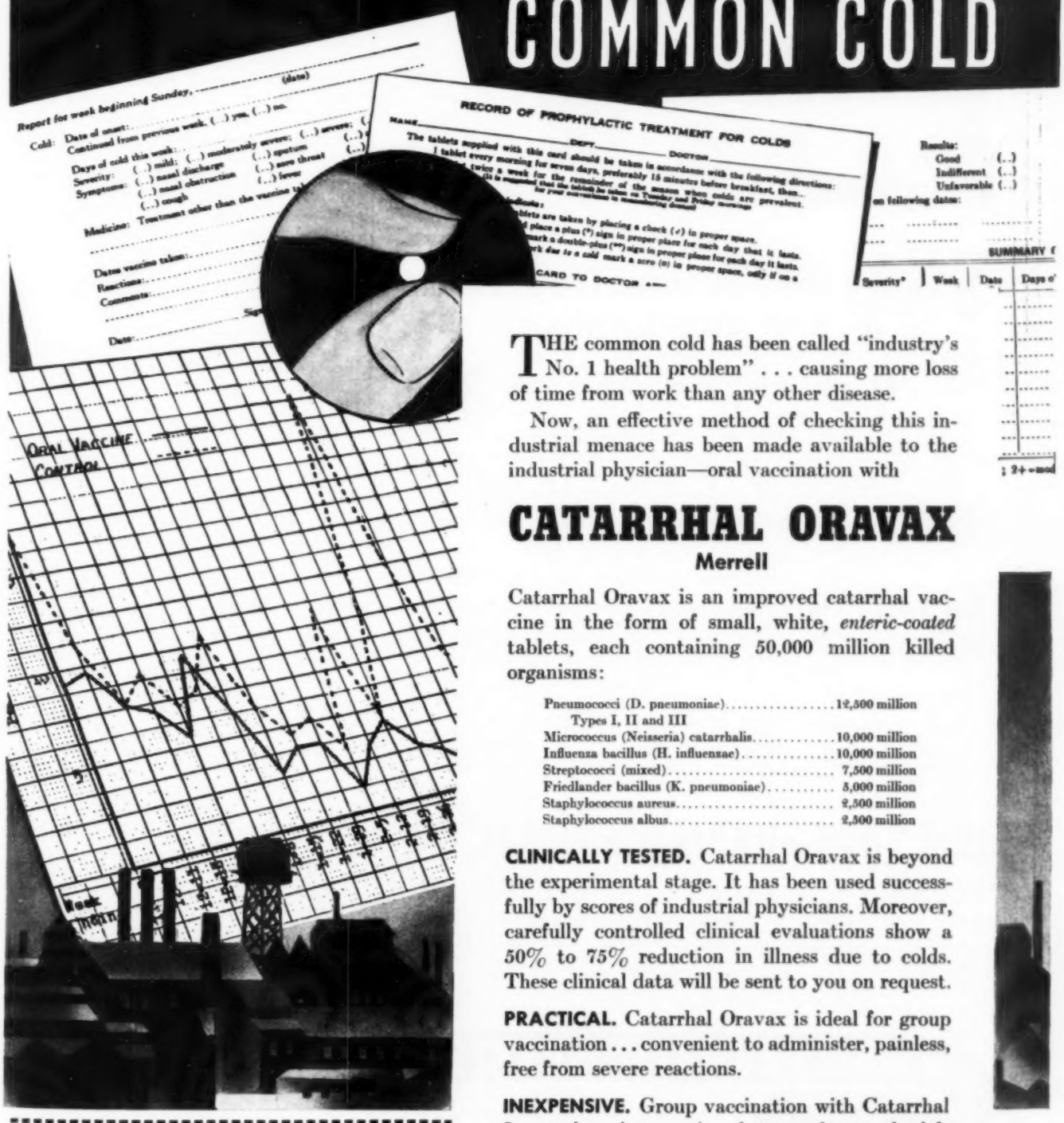
TABLE II

PROPHYLAXIS OF THE COMMON COLD WITH CATARRHAL ORAVAX TABLETS

(Quaker Oats Company, St. Joseph, Missouri)

Worker	Usual annual susceptibility to colds.	Colds experienced the Winter following prophylaxis.
L. B.	chronic	1
B. P.	4	1
M. Z.	6	5
W. M.	3	1
W. P.	3	1
L. W.	5	1
E. F.	6	2
R. H.	6	2
B. P.	6	2
D. W.	chronic	2
A. S.	4	1
F. E.	2	1
M. W.	chronic	1
C. C.	chronic	3
W. M.	4	1
W. C.	5	2
R. O.	chronic	0
C. G.	chronic	1
A. S.	5	1
A. T.	6	2
K. A.	4	2
R. R.	chronic	1
C. S.	3	0
H. P.	4	1
M. S.	6	1
J. S.	6	1
L. J.	2	0
W. C.	4	1
A. R.	5	1
J. V.	chronic	0
W. H.	chronic	3
J. W.	7	2
E. W.	4	1
W. B.	5	2
L. P.	chronic	1
T. G.	3	1
S. W.	4	1
W. P.	6	1
T. H.	4	4
C. B.	2	3

A Clinically Proved Method of Group Vaccination against the COMMON COLD



THE common cold has been called "industry's No. 1 health problem" . . . causing more loss of time from work than any other disease.

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Catarrhal Oravax is an improved catarrhal vaccine in the form of small, white, *enteric-coated* tablets, each containing 50,000 million killed organisms:

Pneumococci (D. pneumoniae) Types I, II and III	12,500 million
Micrococcus (Neisseria) catarrhalis	10,000 million
Influenza bacillus (H. influenzae)	10,000 million
Streptococci (mixed)	7,500 million
Friedlander bacillus (K. pneumoniae)	5,000 million
Staphylococcus aureus	2,500 million
Staphylococcus albus	2,500 million

CLINICALLY TESTED. Catarrhal Oravax is beyond the experimental stage. It has been used successfully by scores of industrial physicians. Moreover, carefully controlled clinical evaluations show a 50% to 75% reduction in illness due to colds. These clinical data will be sent to you on request.

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INEXPENSIVE. Group vaccination with Catarrhal Oravax is so inexpensive that one day on the job which might otherwise be lost will pay for the cost of the individual's vaccination many times over. Your inquiry will bring complete cost figures.

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the midday meal, was chosen mainly because of convenience to the workers, since many of the individuals reported for work at different intervals during the morning. A registered nurse administered the tablets and in this way we had positive proof that the dosage was correct in each instance.

An accurate record, of course, was not obtainable as to the exact number of colds suffered during the previous year by those cooperating in this investigation. For this data, therefore, it was necessary to depend upon the memory of each individual; however, since this factor holds for both the control and the vaccinated group it may be assumed that the values may be comparable for each group and may be used as a basis in the evaluation of the vaccine treatment.

These were not selected cases and this study indicates what practical results might actually be expected in an industrial institution.

A group working under the same conditions was used as a control. The employer furnished the material for this experiment at a total cost of $26.50 or 66c per individual.

Reactions are to be expected following any method of immunization and we found the above mentioned vaccine no exception. About 85% of the individuals complained of some sort of discomfort following ingestion of the tablets. The complaints consisted mostly of muscular aches and nasal congestion. In some instances, headaches were reported. In general, we would say that these reactions were mild compared to the rather severe reactions often experienced after immunization by injection.

In nearly every instance, the individuals in the experimental group who reported colds mentioned the fact that when a cold developed, their symptoms, distress and loss of time were considerably reduced.

Summary

AS IS shown in the Table 1, the average number of colds during the previous seasons was 4.85 in the experimental group. During the Winter following immunization, the average number of colds in the experimental group was 1.45—a decrease of 70.2%.

In the control group, the average number of colds in previous seasons was 4.2. During the experimental period the average number of colds in this group was 3.9—a decrease of 7.2%.

Conclusions

A CONTROLLED study of the effect of oral vaccine on the common cold in a group of 40 industrial workers is reported.

2. Vaccination for the common cold by the oral route is an inexpensive and effective step toward the

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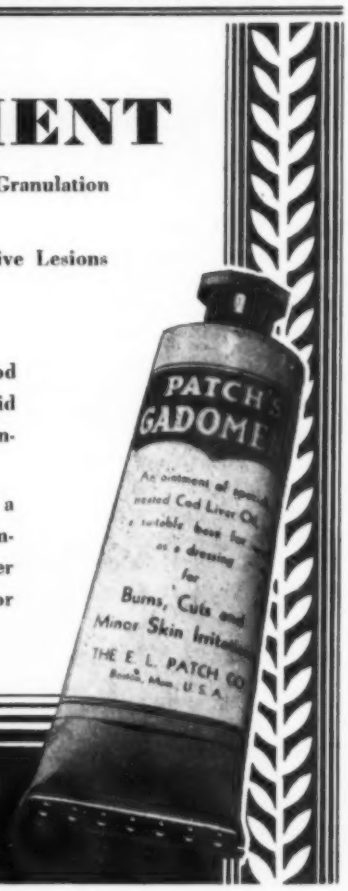

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5. Oral immunization against the common cold is just as effective in the Middle West as it is in the Southern and Eastern sections of the United States.

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Home Hygiene Classes in Industry

GLADYS A. JAHNCKE, R.N.,
Spiegel, Inc., Chicago

AS A PART of the health program of our plant, classes in home hygiene and care of the sick have been made available to women employees.

From *Public Health Nursing,* June, 1940.

Spiegel, Inc., is a Chicago credit mail order house with 5500 employees, of whom 3000 are women. The course was sponsored by the American Red Cross, which contributed the instructor for the classes. The idea was introduced to a group of plant supervisors, department managers, and workers from various departments by the director of classes in the Chicago chapter. The plan was enthusiastically received and the interest of this group soon spread throughout the plant. More than 200 employees registered for the classes, which were divided into groups of 20.

Two schedules were offered for the classes, which are two hours in length: twice a week for six weeks or once a week for 12 weeks. They are held in the assembly room in the plant each afternoon, Monday through Friday, at 4:40 p.m., 10 minutes after the end of the working day. The employees prefer this hour because many live far away from their work and there would be much time lost going home and returning for evening classes.

There are milk-vending machines throughout the plant and many employees avail themselves of this service before going to class at the end of the day.

The American Red Cross textbook is used. Two or more employees may purchase a book and use it together. Demonstration materials are furnished by the local Red Cross chapter, and remain at the plant until the course is completed. Many small items such as medicines, wax paper, spoons, and cups to be used for demonstrations are furnished by members of the class. The Red Cross certificate is awarded to those who successfully complete the course.

That the classes meet a real need is evidenced by the interest and response from the employees. One group of employees wrote:

These employees wish to express their gratitude for the fine instruction they received and feel that they have been more than repaid for the time spent in these classes.

Many individual expressions of appreciation were received, of which this one is typical:

I have a chronically ill relative at home and our home hygiene class has been such a help to me. I have learned how to help handle her more easily and I've learned ways to make her more comfortable.

Another employee stated:

It is surprising what little attention we pay to the importance of a well balanced diet. Our home hygiene course has taught us much about proper food. I feel this was a valuable part of the class work.

The class work is much discussed throughout the plant. The men employees have asked us:

When are you going to give classes

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In this work the anatomy, physiology, and pathology of the thyroid gland are described and brought up to date. Special chapters are devoted to obscure diseases. Etiology of the simple goiter is discussed in connection with prophylaxis and treatment. Malignant goiter is presented under a simplified classification and a new technic for extirpation described. The subject of thyrotoxicosis is covered in a section occupying over half the book and treatment by medical, radiological and surgical methods is described fully. Surgical technic, including pre- and postoperative treatment is given in detail with the aid of drawings of actual operations. By CECIL A. JOLL. 682 pages, 283 illustrations, 24 color plates. Price, $15.00.

STONES and CALCULOUS DISEASE of the URINARY ORGANS

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PRINCIPLES of SURGICAL CARE

In this work, the methods given by Dr. Blalock are known to be successful because they have been proved successful — they are supported by his personal clinical observations. The principles given are general principles in the main with a minimum of detail. The volume deals not only with shock or peripheral circulatory failure, but also with such related problems as dehydrations, hypoproteinemia, avitaminosis, anoxia, pulmonary complications, and intestinal distention. Since their clinical importance extends far beyond the genesis of and prevention and treatment of shock these are treated as separate entities and not just contributing factors. By ALFRED BLALOCK. 308 pages, 13 illustrations. Price, $4.50.

MECHANISM of the LARYNX

In this book special attention is not directed to the study of the human larynx, nor is there an eye to the elucidation of the diseases which attack man. The whole animal kingdom is considered entirely without favor and solely in regard to the normal mechanism. The endeavor has been to describe the origin of the simplest organ and to add on piece by piece a consideration of the modification demanded by complication of life in the animal kingdom. A resume of the structure and function of the human larynx is given and the author takes the stand that the primary function of the organ is not a vocal one, but rather traces reasons for its existence to the demands made on it. By V. E. NEGUS. 528 pages, 160 illustrations, 15 charts. Price, $13.50.

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Physical Examinations of Industrial Workers

PAUL A. BREHM, M.D.,
Supervisor, Industrial Hygiene Unit
State Board of Health, Madison

A PROGRAM of pre-employment physical examinations and periodic reexaminations of workers in all types of industries was recently proposed in Wisconsin.*

This program is of great importance to the practicing physician and for that reason he should become thoroughly familiar with the procedures of the examination and their significance as they apply to the applicant's job or proper placement. It is not the intent here to interpret physical findings that deviate from the so-called normal. The evaluation of the physical fitness of an industrial worker must rest upon the clinical judgment of the examining physician. Such examination must not be taken lightly, because the physician is passing judgment on one who desires to become a wage earner or to continue in that capacity. The utmost care must be exercised in physical examinations of industrial workers to enable the examining physician to render an honest and accurate opinion in every industrial case.

Important Features in Examination

OCCUPATIONAL and medical history. — In making a physical examination, perhaps one of the most important features is a detailed and accurate occupational history. The past jobs and exact occupations should be noted in their order along with the duration of each. This is significant from the standpoint of past environmental exposure and has a direct bearing on any similar exposures which are contemplated at the time of the examination or in the future. Occupational exposures, on the other hand, are valuable guides in the actual examination for the purpose of determining any signs or symptoms of ill effects resulting from such exposures.

A medical history is necessary in any physical examination. In the case of industrial workers, a history of respiratorry diseases is important, particularly pertaining to tuberculosis infection or exposure. The medical history should include diseases that are related to employment where exposures to environmental conditions must be taken into consideration during the ensuing examination. Any diseases or disabilities which may influence the safety of the employee or his co-employees should be determined, as, for example, epilepsy.

If the applicant for a position is a prospective employee, the exact occupation should be designated. This will serve as a guide for subsequent reexamintaions in the event that the occupation is a potentially hazardous one. In the case of an old employee, it is necessary to record the exact occupation and the length of time he was employed. Again, in this regard, potentially hazardous working conditions should be kept in mind by the examining physician.

The present complaints of the applicant may be indications of ill health and should serve as guides during the examination. Substantial loss of weight in any individual, especially over a short period of time, should receive consideration, particularly from the standpoint of active tuberculosis.

VISION AND HEARING.—The test for vision is a very essential portion of a routine examination, notably where an occupation entails potential accident hazards. The examining physician must exercise judgment in cases of defective vision, depending upon the degree of impairment and the occupation of the employee.

Defective hearing is likewise significant as it is related to employment and specifically as it applies to occupations in which defects of this function would render the applicant unsafe, as well as those in the immediate vicinity.

Wisconsin Medical Journal, September, 1940.
* Wisconsin Physical Examination Program, Industrial Commission of Wisconsin, 1939.

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FOCI OF INFECTION.—The examination of the teeth should be carefully made to detect possible foci of infection or other conditions calling for dental care. In the examination of the throat, it is important to note disease of the tonsils, especially from the standpoint of foci of infection, as well as a predisposing factor to frequent upper respiratory infection.

HEART, CHEST, ABDOMEN. — Careful examination of the heart is indicated in all individuals, chiefly those engaged in strenuous occupations. Abnormalities of the heart in such occupations may mean the safety of several individuals. The interpretation of blood pressure findings must receive careful consideration by the examining physician. An elevated blood pressure associated with either heart or kidney damage or both is significant in any occupation that calls for physical strain or one that may endanger the safety of others. Slight elevations of blood pressure unassociated with other pathological findings may be no more indicative than mere nervous tension on the part of the applicant during the time of examination.

Examination of chest and lungs is essential in detecting signs of disease, mainly tuberculosis, or indications of other chronic diseases as related to hazardous occupations. Some employers do not include x-ray studies of the chest in a routine examination and, in this event, the examining physician must exercise special caution in his conduct of the chest examination. Similar caution is indicated, too, in the case of other employers, and in particular in those plants where there is no specific pulmonary hazard, who, instead of providing for the approved procedure of making a flat x-ray plate of the chest for study, prefer, in order to lower examination costs, to have the physician conduct either a fluoroscopic ex-

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amination or do tuberculin tests, limiting x-ray study to the positive reactors.

The abdomen may disclose signs of pathological conditions which may influence the health of the employee or his proper placement. In occupations where exposure to materials such as solvents may result in liver damage, the examination should emphasize this region. Additional diagnostic procedures in such cases may be indicated. Hernias of all types should be carefully detected because of their importance in jobs calling for any physical strain.

BACK, SKIN, EXTREMITIES.—Careful examination of the back should be made on all employees, principally those engaged in strenuous work. The range of motion and function should be taken into consideration. Evidence of disease or deformity must be weighed in the light of the physical requirements necessary for a particular occupation. The inattention devoted to this region may result in confusion and miscarriage of justice in the event of a future compensation claim.

The skin of the entire body should be examined to detect evidence of any existing lesions. The presence of skin lesions is influential in the placement of an applicant in an occupation where dermatitis may result from exposure to materials incident to the manufacturing process.

Extremities should be examined for any evidence of muscle or nerve impairment. Arthritis changes of joints and limitations of motion call for exercise of the physician's judgment as to whether the functional capacity is sufficient for a particular job.

Examination of the reflexes should include more than merely an elicitation of the knee jerk — they should be sufficiently complete to detect signs of dysfunction. The ill effects from occupational exposures to some toxic substances may be manifest in abnormal reflex actions and it is necessary for the physciian to bear this fact in mind while examining an employee with a known exposure of this kind.

Varicose veins should be noted in regard to their degree and location and considered from the aspect of their influence on various types of jobs which might aggravate the condition or result in prolonged disability in the event of an accidental injury.

ENLARGED GLANDS, ELEVATION TEMPERATURE. — Glandular enlargement, particularly lymph and thyroid, should be given careful study to determine any ill effects upon the general health and efficiency of the worker.

Any elevation of temperature should always call for an explanation.

Laboratory Tests

BY MEANS of a Tallqvist hemoglobin determination and a quick cover-glass smear an adequate indication may be obtained regarding the condition of the blood. Abnormal findings by means of these two tests should be followed by a more detailed blood examination.

Albumin and sugar tests of the urine are sufficient for the purpose of routine examinations. The finding of either albumin or sugar in the urine is an indication for observation by the applicant's physician.

Blood should be taken for the diagnosis of syphilis. The importance of this diagnostic procedure for industrial workers cannot be over emphasized. Syphilis in the infectious stages should exclude an employee from work until adequate treatment has rendered him non-infectious. The only other reason for exclusion of a syphilitic person from employment is involvement of the central nervous system to the extent that the safety of the individual and his co-workers is imperiled.

X-ray films of the chest, in the course of a physical examination of industrial workers, are necessary for the accurate diagnosis of tuberculosis lesions and should be taken, if possible, without regard to occupational exposure. The exclusion of a worker with active tuberculosis from contact

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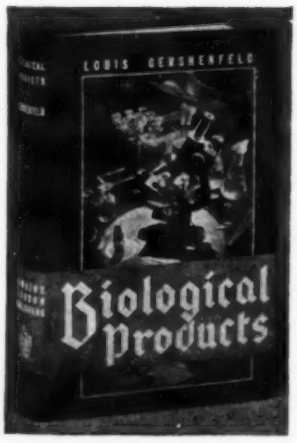

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with other employees in a work room area is sufficient justification for the additional cost involved on the part of any industry. The diagnostic value of x-ray films in tuberculosis control is dependent upon the qualifications of the clinicians making the interpretations.

Abnormal findings in the course of a physical examination may indicate that special procedures are necessary. If these are made, the results should be recorded on the examination form. Sputum tests and blood sedimentation rates are not made in a routine examination unless questionable findings indicate their necessity. Also, in certain industries where specific hazards are encountered, it may be necessary to employ special diagnostic technic, as, for example, careful examination of the blood in those individuals who may be exposed to lead compounds.

The duties of the examining physicians in making the above examinations do not cease at the end of a careful investigation or the reporting of the results of the examination to the employer of the employee. Rather, it should be one of the primary duties of a physician, when called upon by an industry to examine the workmen, to acquaint himself thoroughly with the manufacturing processes in that plant. He should know the physical requirements of the jobs and the materials to which the workers are exposed and he should know the ill effects which might result from exposure to excessive concentrations of such materials. Only by means of such a working knowledge can any examining physician be on the alert for early manifestations of diseases arising in the course of employment and be able to render a just opinion as to the employability or placement of the individual.

Pacific Coast News

AT THE last meeting of the California State Medical Association, DR. DONALD CASS, of Los Angeles, was appointed Chairman of the Committee on Industrial Practice. The other member are DR. MORTON R. GIBBONS, of San Francisco, and DR. GEORGE H. SANDERSON, of Stockton, California.

On September 16, 17, 18, and 19, 1940, on the program of The Department of Health Officers League of California Cities to be held at San Diego will appear DR. JOHN P. RUSSEL, Chief of the Industrial Hygiene Service of the California State Department of Health, who speaks on "Industrial Hygiene and the National Defense Program."

Two preliminary meetings have taken place in preparation for the organization of a Component Society of the American Association of Industrial Physicians and Surgeons, to be known as the PACIFIC COAST SOCIETY OF INDUSTRIAL MEDICINE AND SURGERY. The first meeting is to be held at Del Monte Hotel, at Monterey, California, the day preceeding the California State Medical Association. Plans are now being made for a scientific program and invitations will be sent to industrial physicians and surgeons to become members. The temporary secretary is DR. RALPH CHRISTOPHER LEGGO, Surgeon of the California Hawaiian Sugar Refining Corporation, at Crockett, California.

The Los Angeles City Health Department, recognizing the growing importance of industrial medicine, have instituted a Division of Industrial Hygiene in their department and have appointed DR. A. V. NASATER as Director.

The University of California Extension Division at San Francisco, commencing September 12, is offering a course in Industrial Hygiene for nurses. DR. ROBERT T. LEGGE. Professor of Hygiene, of Berkeley, is the instructor.

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Nutritional Protection During Senescence

DURING senescence, many conditions are prevalent which tend to prevent the adequate utilization of a well balanced diet. Degenerative processes, voluntary or required dietary restrictions and organic disease, especially if it occurs in the gastro-intestinal tract, all play an important role in interfering with proper digestion, absorption and assimilation. In this manner, they pave the way for nutritional deficiency states.

It is generally recognized that an adequate, energy-producing and nutritional protective diet is an essential requirement during old age. However, since an ample dietary regimen is usually not well tolerated by this type of individual, a fortified food drink was used as a supplement to their regular therapy. The purpose of this supplementary feeding was two-fold; first to supply essential vitamin mineral elements and secondarily. by using it as a flavoring agent to act as an incentive to these individuals to drink the required amounts of milk.

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In conclusion, it may be said that the fortified food drink (Cocomalt) by itself may have limited therapeutic value. However, when used as a supplement to the regular necessary treatment, it is no doubt a valuable method in supplying essential vitamin-mineral elements in a pleasant form.

HARRY BAROWSKY, M.D., in *Medical Record*, August 21, 1940.

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The Editorial Committee will exercise its best judgment in selecting for publication the material which presents most exactly the factors affecting industrial health and developments for control of potentially injurious exposures. The editors may not concur in opinions expressed by the authors but will endeavor to assure authenticity of fact.

The Science, the Law and the Economics of Industrial Health

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Toxic Logic

TOSS the dust counters into a closet, lock the door and lose the key." "Dust is present, therefore a hazard exists." "This man has worked here the last 40 years without a sick day in his life: the place must be safe." "Carbon tetrachloride is less toxic than gasoline." "Gasoline is less toxic than carbon tetrachloride." "Air analyses don't mean a thing—the conditions change every day." "Toxic limits are valueless—they are all based on animals."

These and many another loose statement have been made and repeated on every side. The closer to the truth the statement is—without actually hitting the bull's eye—the more misleading the impression is apt to be. A general assertion about a scientific fact can often appear logical, even though it is essentially erroneous, by presenting only a portion of the whole situation. By the application of sound common sense to the use of toxic limits for the interpretation of air analyses, the industrial hygienist can utilize a valuable tool for the more effective and more economic control of occupational disease exposures.

A well-rounded presentation of the value of toxic limits, their practical purpose and the limitations of their use has been prepared for this issue by one of the world's most eminent industrial hygienists. His views are based on a wealth of experience in the field and an extensive knowledge of the literature. He is in excellent position to evaluate the reliability of toxic limits which have so far been published, as most of the actual experimental work has been done on them during the period of his active participation in industrial hygiene. Where the toxic limits of a number of organic solvents were determined on the basis of several hours' experiment on a small number of guinea pigs during the first decade of the century, he visualizes the results, not as figures on a published table to be blindly accepted, but as a group of experiments, the findings of which were published in detail as current literature earlier in his career.

The fallacy of the statement that "carbon tetrachloride is more toxic than gasoline" is that this does not apply to prolonged exposure but was based on brief animal experiment. To be sure, the literature states that only 2.2% of gasoline vapor in air will cause death whereas 4.8% of carbon tetrachloride is required to produce this result, but these findings are based on the concentration which kills a cat in 30 minutes. Where there is prolonged exposure, such as is contemplated in industrial occupation, it has been shown that carbon tetrachloride concentrations should be kept appreciably below those considered safe for gasoline vapor.

DR. TELEKY's observations are the results of his own experiences and may not represent exactly the views of all our readers whose contact with the occupational disease situation may be from a different angle. The point of view of those who approach industrial hygiene in its various phases will be welcomed in connection with the conclusions expressed in DR. TELEKY's paper.

A real endeavor should be made to utilize this tool further. The difficulty of not having data on exposures over long periods of a worker's employment, referred to by DR. TELEKY, should be provided for ahead of time by keeping on record average and representative exposures at potentially hazardous occupations from month to month or year to year depending on the nature of the operation.

An increasing number of industrial concerns and those engaged in mining are obtaining and collating just such data. Not only are these data of value in checking exposures, but they will also afford more exact and reliable information on which to base toxic limits.

The development of methods for the quick and easy determination of injurious materials in air is a long step in the right direction. The caution referred to by DR. TELEKY is to be observed, but it is only by use of methods which short-cut laborious laboratory procedures that we can hope for the mass of data required for positive conclusions.

Appreciate the value of toxic limits but, in making use of them, do not fail to observe their limitations.

We commend DR. TELEKY's paper to your critical attention with the thought that for the full utilization of the toxic limit, one must apply the logic of practical common sense.

Economy in the Design of Exhaust Systems

THEODORE HATCH,*
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THE cost of controlling the health hazards associated with manufacturing processes may properly be regarded as a basic part of the production cost and is generally accepted as such today. This is in contrast to an earlier view that such costs represented an unnecessary addition to plant overhead. Too frequently, control equipment was installed in the past simply to satisfy some regulatory body and the main criterion was cheapness, without regard to efficiency. Little attention was paid to the essential engineering details of design and, as a consequence, money was wasted without getting the desired results. In general, the low initial cost was found to be offset by high operating cost due to excessive power consumption which made the equipment too costly to operate. The application of sound engineering principles to the design of control equipment however has demonstrated that good results can be obtained and, at the same time, that relatively great savings can be effected in the installation and operating costs. It is the purpose of the present paper to discuss in particular economy in the design of exhaust ventilating systems but the value of other more fundamental control measures must not be overlooked. For example, the substitution of a non-toxic substance for hazardous material or changing the process so as to reduce the rate of dispersion of the offending substance or the use of mechanized processes in place of manual operations are all basic improvements which may eliminate the need for ventilation or, at least, reduce the work which the ventilating system is required to do. The extent to which such changes can be made will depend upon the cost as compared with the savings effected. In certain cases where the hazard is a serious one, the substitution of a non-toxic material may be justified in spite of the higher direct cost.

The cost of operating an exhaust system includes the carrying charges on the initial investment, the depreciation and maintenance charges, the power cost and the extra heating expense imposed by the increased ventilation. In some cases there will be an additional labor charge because of the interference with production by the ventilating apparatus. Much can be done to reduce the costs of maintenance, power and heat through proper design and construction of the exhaust system. In many cases these savings can be effected without materially increasing the initial cost of the system and such savings therefore represent clear gain. In others, however, operating costs can be reduced only by increasing the initial expenditure for the control equipment and the most economical system must be determined by balancing increased carrying charges against savings. It is not the purpose of the present paper to analyze actual costs but rather to consider a number of

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* Now Associate Professor of Industrial Hygiene, University of Pennsylvania. Presented at the First Annual Meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION, New York, June 5, 1940.

good engineering practices, the application of which will reduce the cost of operating exhaust systems.

Power Consumption

THE power consumed by an exhaust system is determined by the product of the rate of air flow and the overall resistance of the system and is further influenced by the efficiency of the fan, thus

$$BHP = \frac{Qh}{6350\, E_{fan}}$$

Where BHP=brake horsepower
Q=rate of air flow, cfm
h=overall static resistance, ins.w.g.
E_{fan}=static efficiency of fan

Anything that can be done to reduce Q or h or increase E_{fan} reduces the power consumption. In general, the horsepower required is not great for a single system and, since the overall resistance does not exceed a few inches of water, a quarter of an inch of extra resistance here or there or the use of a few hundred cfm excess air flow is not regarded as serious. Relatively, however, these small extra values may be of great importance and proper attention to details will result in a reduction in power consumption of 25% or even more, which for a large establishment, becomes a major saving. In the small plant it is of greater relative importance although as an absolute value it will not be striking. For industry as a whole, it becomes of very great consequence. In New York State, for example, approximately 10 million cfm of exhaust ventilation was installed in 1939, requiring nearly 10,000 H.P. An average saving of 25% would reduce the power cost many thousands of dollars.

Design Requirements for Minimum Rate of Air Flow

THE minimum value of Q for a particular exhaust system equals the sum of the rates of air flow required for the several hoods. A fan capacity in excess of this must be provided, however, to make up for the lack of balance in the system which requires that the rate of air flow through hoods near the fan be increased above the minimum in order to maintain the necessary flow through the remote hoods. In a poorly constructed exhaust system additional fan capacity must also be provided to counteract the leakage at pipe joints.

Thus, the design requirements for minimum rate of air flow are:

1. High hood efficiency (minimum rate of air flow per hood)
2. A balanced system
3. Tight construction

In spite of the fact that empirical methods are generally employed in hood design it is possible to consider hood performance requirements from a fundamental standpoint and from this to develop many useful ideas, the application of which lead to improved design and reduced air flow requirements.

The function of an exhaust hood is to create in the zone of generation or release of the contaminant air velocities toward the exhaust opening of sufficient magnitude to overcome the forces tending to disperse the substance into the atmosphere and thus to draw it into the exhaust system. It follows that the hood velocity requirement will be decreased by any action which reduces the magnitude of the dispersive forces.

With respect to the forces of dispersion, I have pointed out elsewhere[1] that dynamic dispersion of dusts, that is, dispersion by virtue of the kinetic energy of the dust particles themselves, is not of consequence except in the case of relatively large particles ($> 20\mu$) and that microscopic particles ($<10\mu$) are scattered only by virtue of the movement of the air in which they are suspended.

Thus, the control of fine dust is not a matter of removing the dust itself from the air but rather of collecting the *dust-laden air*. This is also the case with vapors and gases. Hence, it is the air movement in the zone of generation of the contaminant, created by the process or otherwise, which must be overcome by the exhaust hood.

There are many sources of disturbing air motion which can be attacked directly with beneficial results. These have been summarized elsewhere[2] and only a few examples will be cited here to illustrate their importance in hood design.

1. Air is displaced when crushed rock falls through a chute into a container or onto a belt. the air movement taking place in the direction of flow of the material. The amount of air displaced varies with the distance of fall, particle size of the rock and the rate of flow of the material, and also depends upon the resistance to air flow imposed by the system. In one case, actual measurements showed that the rate of air displacement amounted to fifty times the volumetric rate of flow of rock with outward air velocities from the container being filled of the order of 300 fpm in one case and 1100 fpm in another.[3] The rate of air displacement can be cut down by reducing the area of openings through which air enters the system, by breaking up the fall of the rock or by keeping the lower part of chute full of rock. A surge chamber at the bottom of a chute just above a conveyor belt with a floating gate, as shown in Fig. 1, will practically eliminate the outward escape of air and reduce to a minimum the dispersion of dust.

2. Air motion is created by rotating and reciprocating machine members on the process itself or on nearby machinery. Proper shielding of such parts will reduce the rate of air displacement. A striking

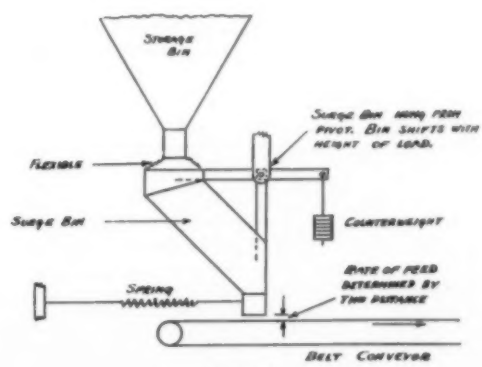

FIG. 1.
Dustless Ore Chute

example of this source of air motion was seen in a cable covering and impregnating machine. The rotating spool for applying the spiral cover was located adjacent to the hot asphalt impregnating tank and the strong fan action of the revolving wheel dissipated the asphalt fumes with considerable velocity. A shield between the two greatly reduced the hood velocity requirements.

Another example, familiar to all, is the common grinding wheel which actually blows air out of the exhaust hood in spite of the strong inward suction. A scalping vane, as shown in Fig. 2, was found to reduce the adverse fan action greatly, although its practical application to grinding wheels is doubtful, owing to the need for frequent adjustment as the wheel wears.

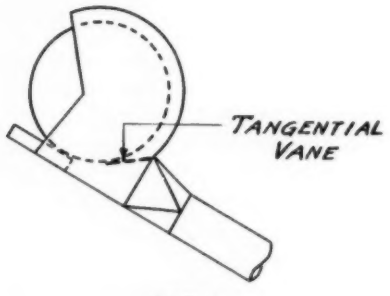

FIG. 2.
Scalping Vane to Improve Action of Gringing Wheel Hood

Convection currents set up by a hot process may increase the ventilation requirements. In a study of steam tank ventilation, for example, steam-laden air was found to escape out from under a canopy hood over the tank when the ventilating rate was under 800 cfm although the volume of steam generated was less than 50 cfm. Thus, a vertical flow of room air of some 15 times the rate of steam generation was created immediately around the tank. Suitable insulation of the tank would have reduced the strength of the convection currents greatly and with it the required ventilation rate.

3. Leaky compressed-air operated tools are frequent offenders and may create relatively great air velocities in the zone of dust generation. The leakage of air out around the shank of the pneumatic granite surfacing tool sets up a strong air blast onto the stone immediately adjacent to the cutting area and thus scatters the dust. A baffle disk on the shank of the tool eliminates this, or better, a series of vents around the chuck chamber itself directs the air away at a point well above the stone thus decreasing the velocity of dust dispersion.

4. The hood velocity required to overcome the dispersing force can be reduced in certain instances by increasing the distance over which the opposing velocity works (since work equals force times distance). Thus, spray booths should have sufficient depth to minimize the escape of spray materials due to rebound. Inspection openings in the walls of enclosures may be provided with side baffles to make a tunnel-like opening instead of a simple orifice, as shown in Fig. 3. Theoretically, if the distance over which the controlling velocity acts is doubled, the velocity itself may be reduced some 30% for equally effective control.

5. Uncontrolled drafts and convection currents in the work shop may produce needless dispersion of the contaminant. In a study of degreasing tanks[4] the need for eliminating such disturbing currents was clearly shown. In this connection, special care may be required in locating intakes for the entrance of make-up air into the room to minimize disturbance.

The foregoing examples are illustrative of the measures that can be taken to reduce the work to be done by exhaust hoods. The controlling velocity finally required is preferably determined by direct experimentation which yields a correlation between the rate of ventilation and the concentration of contaminant in the breathing zone. The expense involved in such studies is not great compared with the benefits derived therefrom and only by means of data thus collected can hood design be placed upon a fundamental basis. The volume of literature on this subject is increasing but much remains to be done by the various organizations concerned. It is especially recommended that the ventilation requirements of particular machines be investigated by the manufacturers themselves who will then be in a position to make definite recommendations to the purchaser.

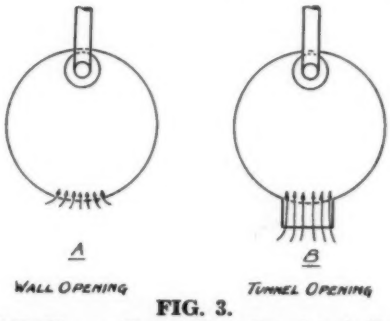

FIG. 3.
Tunnel Baffles on Inspection Opening to Increase Distance over which Velocity Acts

Hood Efficiency

THE rate of air flow through the exhaust hood required to develop the controlling velocity in the zone of generation or release of the contaminant depends upon the degree of enclosure around the process, or the closeness of the hood face to the area over which the contaminant is released and the opportunity for air to be drawn from areas other than the area of contamination. These factors combine to determine the aerodynamic efficiency of the hood which may be defined as the percentage ratio of the volume of air which does useful work to the total volume of air flowing into the hood. Although it is not possible to describe the air flow characteristics of complicated hoods, much useful information may be had from a study of the velocity contours of simple, unobstructed suction openings which have been described fully by DallaValle.[5]

Of greatest importance among the flow characteristics is the relation between velocity and center line distance from the hood face which may be defined approximately by the equation

$$V=\frac{0.1Q}{X^2+0.1A}$$

Where V=velocity at X distance out along center line, fpm
Q=rate of air flow through hood, cfm
X=distance out, feet
A=area of hood opening, sq.ft.

According to this equation, the velocity varies inversely with the square of the distance, which emphasizes the need for bringing the hood face as close as possible to the point of work. For example, Q may be quartered for a given controlling

velocity if the distance X to the point of generation of the contaminant is reduced by one-half.

A second factor to be noted is the relative unimportance of the velocity at the face of the hood which is found to have little influence upon the velocity any appreciable distance in front of the hood. It is the rate of air flow and not the face velocity which is important. From this, it may be said that for a given value of Q the face velocity should be made as low as possible by increasing A, the area of the hood opening. This has the advantage of establishing flat contours directly in front of the hood, and improves the hood efficiency by increasing the percentage of useful air flow.

The value of side baffles to reduce the air flow from ineffective areas back of the hood face has been demonstrated not only by studies of the velocity contours but also by direct experiment. Reductions in air flow of 25% or more have been effected through the proper use of baffles. Internal baffles, as illustrated in Fig. 4 are said to improve the flow pattern in the same way. They are also effective in decreasing the turbulence and eddying at the hood edge which may decrease hood efficiency[6] and are of value in controlling rebound from within the hood, as in a spray booth.[7]

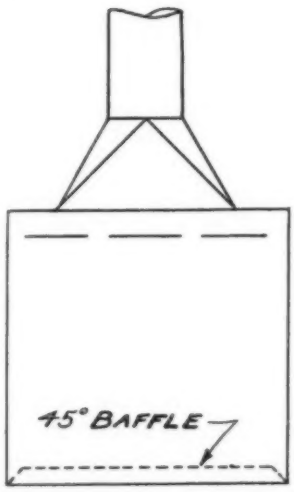

FIG. 4.
Internal Baffles on Spray Booth

These flow characteristics apply to the free-hanging hood. They are also applicable to lateral hoods and other semi-open conditions. In general, however, the objective in hood design is to provide an effective enclosure around the process and to such hoods the above laws do not apply. For high hood efficiency the enclosure must be as complete as possible and the controlling openings should be kept away from the major source of disturbance within the enclosure. In this way the unequal pressures that are built up will not create a high outward velocity through the openings.

The foregoing is not a complete summary of the design requirements for high hood efficiency, but it serves to direct attention to the fundamental approach through which savings may be effected.

Design of Exhaust Systems for Balanced Flow

IN A balanced exhaust system the rate of air flow through each hood equals or approximates the flow required by the hood design with no significant increase in the air flow rate for the hoods near the fan. In the common high-velocity system, however, the pipe sizes are simply calculated to give a uniform velocity (the so-called area-load method of design) throughout the system. The unequal distance of travel from the fan to the various hoods with a consequent difference in resistance leads to an unbalanced distribution of flow. The increase in the total rate of air flow required to insure the minimum flow in the remote hoods amounts in some cases to 30% or more. In order to improve the balance, an effort is made to provide a pipe layout symmetrical about the fan with not unusually long branch pipes and thus to equalize resistance. Blast gates may be used for a final adjustment although they cannot be depended upon permanently owing to wear and improper manipulation. It is a relatively simple matter to secure a balanced design in a system used for the removal of gases since the pipe velocities are unimportant and can be adjusted as required to compensate for the differences in resistance. In a dust exhaust system, on the other hand, the velocity cannot be reduced below that required for transporting the collected dust and all adjustments in pipe velocities must therefore be made in the range above this limit.

The following steps should be followed so far as possible to approximate a balanced design:

1. Provide for a symmetrical layout with minimum variation in the length of branch pipes and the shortest possible run of main pipe. Two or more systems may sometimes be employed profitably in place of one large system to give greater symmetry and balance.
2. Employ a lower velocity in the main exhaust pipe than in the branches. The effect of this is to make the branch pipe resistances relatively great as compared with the header resistance and thus to minimize the effect of the greater distance of travel to the remote hoods. It is common practice to increase the main pipe area 20% over the "area-load" for this reason and the same practice may be applied to long branch pipes.[8]
3. Increase the velocity in branch pipes near the fan by reducing branch pipe diameters and thus increase the resistance to compensate for the lesser distance of travel to the fan. This can be done by means of simple hydraulic equations of flow.[2] The effect is the same as that given by adjustable blast gates but the method is to be preferred owing to the fact that the adjustments are not subject to further manipulation and will not wear away.

The unequal resistance in the various parts of an exhaust system results primarily from the fact that high pipe velocities are required for transporting the collected material. The most effective design for balanced flow therefore is one that does not make use of pneumatic conveying of the collected material. No branch or main pipes are used and the dust falls by gravity into a low-velocity plenum chamber from which it is removed by a mechanical conveyor. Since the negative pressure is practically uniform throughout the chamber, the rate of flow through each hood is dependent only upon the size of the opening. This method of design has many other advantages[9] which justify its use in spite of the higher initial cost as compared with that of a conventional high-velocity system.

Design Requirements for Low Resistance

THE overall resistance of an exhaust system is the sum of the pipe friction and the losses at the entrance to hoods, in elbows, pipe junctions, transition pieces, through air-cleaning equipment, the terminal discharge and other parts of the system. The resistance set up by these several parts varies with the square of the air velocity and it follows, therefore, that the greatest saving in resistance is given by reducing the pipe velocity. Dropping from 4000 to 3000 fpm, for example, will lower the resistance approximately 50%. Vapors and gases are conveyed at any velocity and the optimum value is determined by striking the economic balance between the cost of the piping and other construction costs and power saving. Standard equations have been developed for this purpose.[10] In dust exhaust systems, greatest savings are effected by eliminating the need for pneumatic conveying.

The equipment has an overall resistance of approximately ⅜" as compared with ⅞" in a conventional system for the same work and the power consumption is only 3 HP whereas it was 22 HP in an earlier high-velocity system which it replaced.

Even in high-velocity systems much can be done to save power through more careful consideration of the transporting velocity requirements. It is common practice to use a value of 4000 fpm for all dusts without regard to particle size, specific gravity or loading and to make no distinction between vertical and horizontal conveying. Velocities of this magnitude and even higher are required for certain materials but others are conveyed successfully at much lower speeds. Laboratory tests indicate that dried stone dust of the size encountered in industry can be moved at 2000 fpm and velocities of this magnitude are known to give satisfactory performance in several industrial installations. On the other hand, laboratory and field experience indicate that for wet material the velocity must be increased greatly. Above a certain moisture content there is no practical velocity that will prevent settlement. Fine wood dust, as generated in a cabinet shop, is conveyed successfully at 2000 fpm whereas heavy wet wood cuttings require a velocity of 4000 fpm or more. It is not my intention to recommend the general use of lower velocities than are now employed and even required by codes but simply to point out that it can be done safely in many instances with marked savings in power. There is need for a critical study of past experience in this connection and a more basic evaluation of the problem in the interest of economy and good engineering practice.

The design of piping and fittings for low resistance follows well-known lines. Hood losses are minimized by providing stream-line connections to the branch pipes. An increase in the hood coefficient from 0.7 to 0.95, which is wholly practical in many instances, decreases the entrance loss from 1.0 VP to 0.11 VP, an improvement of 90%. Elbows are constructed of five or more pieces or even stamped out to form a continuous curve and are given a centerline radius of 1.5 to 2.0 diameters, in order to minimize the elbow resistance. Junctions of branch with main pipes should be made at a gradual angle and preferably in streamline form. The junction loss for an angle of 15° is only 40% of the loss for an angle of 45°. Enlargement sections must be tapered rather than abrupt and for best results should have an included angle of 7°. Entrance and outlet pipes to filter houses should be provided with tapered transition pieces rather than abrupt connections. This reduces the losses at these points approximately 50%.

A badly designed weather cap introduces an unnecessary resistance which is greatly reduced through proper design. The use of a discharge chimney is recommended to recover part of the discharge velocity pressure. A saving of ½" or more, representing, in some cases, 10% of the overall resistance of the system, can be affected by this means alone.

The air-cleaning equipment accounts for a major portion of the system resistance and its design and capacity must therefore receive careful consideration. In the case of cyclone collectors, many special designs have been developed which offer low resistance but one finds frequently that dust collecting efficiency has been sacrificed in order to save power. This must be kept in mind when selecting a collector of the cyclone type. For the common type, the optimum capacity is obtained with an inlet velocity of 4000 fpm. For capacities above this value the resistance is greatly increased without improving the efficiency while the saving in resistance at lower capacities is offset by reduced efficiency. In the case of cloth filter collectors, the resistance is directly related to the ratio of capacity to cloth area and the latter must be carefully determined for each installation if high resistance is to be avoided. Mathematically, the resistance varies with the square of the filtering velocity and directly with the loading and resistance coefficient of the dust[11] according to the following equation:

$$h = \frac{rLtV^2}{7000}$$

Where h = increase in filter resistance, ins., w.g.
r = coefficient of resistance of the dust, which varies markedly with particle size and porosity
L = dust loading, grains per cu.ft.
t = time of operation since last cleaning, minutes

This equation has limited application to filter design at the present time owing to lack of data pertaining to L and r. Its greater use is to be recommended, however, in the interest of power saving on the one hand and optimum filter cost on the other as demonstrated by Hemeon.[12]

The manufacturers of air-cleaning equipment, through their experience, are prepared to make specific recommendations with respect to the type and size of equipment required for each installation.

Fan Selection

THE selection of the most efficient fan with respect to type, size and speed depends upon the capacity and static pressure to be developed

and also upon the nature of the material to be handled. The leading fan manufacturers have developed efficient fans for a great variety of duties and are prepared to make specific recommendations for every installation. Full use of their service will result in significant power savings.

Summary

THE cost of controlling atmospheric contaminants released by manufacturing processes by means of exhaust ventilation includes the carrying charges on the initial investment, maintenance costs, the cost of power and extra heat, and in some cases, an extra labor charge resulting from interference with production by the control equipment. The most economical design of an exhaust system is one which balances the carrying charges against the operating costs to give a minimum value. The present paper considers the various design factors which together determine the power consumption and indicates a number of measures which can be taken to reduce the power charge. These are summarized as follows:

1. Reduce the magnitude of the forces of dispersion of the contaminant, thus decreasing the work to be done by the ventilating system.
2. Provide exhaust hoods of the highest aerodynamic efficiency.
3. Lay out the exhaust piping to give a balanced system.
4. Employ the lowest possible *safe* transporting velocity in the pipes or better, provide means other than pneumatic conveying for the removal of the collected matter.
5. Design hood entrances, elbows, junctions, enlargement sections and other pipe details for minimum resistance.
6. Select air-cleaning equipment of sufficient capacity to insure an economical resistance.

Attention to these and other details will reduce power costs 25% and even more in many cases and will insure longer life and lower maintenance costs. In certain cases these savings will represent clear gain. In others they will more than offset the increased initial cost. Their application requires the use of more fundamental engineering concepts however than are now applied to the design of exhaust systems generally.

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Report of Industrial Hygiene Session

—National Battery Manufacturers Association—

Reviewed by
EUGENE L. WALSH, M.D.

THIS privately printed brochure,[1] which has been previously announced,[2] contains in its first 78 pages the individual papers of the contributors. A supplement of 15 pages presents a verbatim report of the minutes of the Special Medical and Engineering Section.

Each author covers certain specific phases of the lead hazard in the storage battery plant. Obviously this method produces some repetition which is not undesirable, but rather serves to emphasize the need for coordination in terminology and methods in the various laboratory procedures in use today.

The assembled papers cover quite completely the industrial hygiene aspects of the lead problem in one industry. The engineering and medical problems are related in a report of a United States Public Health Service survey of the storage battery industry. The problems arising in the smaller plants are given in other papers. Safety problems receive due consideration and protective devices are very thoroughly considered, both with respect to the individual worker's protection and engineering protective devices for the manufacturing process. The medical and chemical laboratory aids used by the industrial hygienist in the diagnosis and control of lead hazards and intoxications is critically discussed by one capable authority. This topic receives the attention of others, also.

In the second section the organizers of this symposium have attempted, by the informal round table discussion method, to clarify a few of the controversial problems presented in Part I. In general this has been well done and in detail covers some of the more practical phases. Among the subjects discussed are the following: Methods of sampling air for analysis; limits of allowable lead concentrations; modes of dust abatement and worker protection; correlations and recommended practice for laboratory procedure, particularly with reference to stipple cells; the problem of acid fume exposure; and others.

The industrial lead problem is approached from the manufacturer, insurance carrier, personnel

1. Report of Industrial Hygiene Sessions of the 15th Annual Convention. Chicago, October 24-25, 1939. Released by National Battery Manufacturers Association, Akron, Ohio, 1940. $2.00 a copy.
2. INDUSTRIAL MEDICINE, Industrial Hygiene Section, Vol 1, p. 42, July, 1940.

and industrial relations, compensation, employe and medical attitude in the various papers.

This volume is an authoritative practical epitome of the lead problem in the storage battery industry. It has been written by a well-chosen staff of men with wide practical experience. This review may be read with profit by the physician, engineer or executive interested in this industrial hazard.

Glycerol

—Behavior in the Animal Organism—
(A Review of the Literature)

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Kettering Laboratory of Applied Physiology,
University of Cincinnati,
Cincinnati, Ohio

GLYCEROL finds wide application in pharmaceutical preparations. In fact, according to Lesser and Murphy,[38] it is probably used more frequently in prescriptions than any other substance except water. Any toxic properties which it may possess are therefore of considerable importance.

Data on the lethal and sublethal concentrations of glycerol obtained from the literature are summarized in the table.

There are only two cases on record in which glycerol has been known to produce poisoning; these were in children. The first child, two and a half years old, took 300 gm. orally; loss of consciousness followed, but the child recovered after its stomach was washed out. The second case was fatal, but unfortunately neither dose nor mode of administration is known.[31]

The ingestion of 30 cc. of glycerol three times daily over a period of 50 days by normal human subjects was found to be harmless,[29] but subcutaneous or intravenous injection of glycerol extracts may be followed by a local inflammatory reaction or by edema, while the injection of a 50% glycerol-broth culture of human tubercle bacilli in tuberculin-positive patients caused a skin reaction in many instances.[21,24]

Physical and Chemical Properties

GLYCEROL (mol. wt. 92.06) is the simplest alcohol containing three hydroxyl groups:

$$\begin{array}{l} CH_2OH \\ | \\ CHOH \\ | \\ CH_2OH \end{array}$$

It is a viscous, colorless, odorless liquid with a neutral reaction and a sweetish taste; the specific gravity is 1.265 at 15°C.

It is miscible in all proportions with water, alcohol and chloroform but immiscible with ether, fats and oils.

Glycerol usually contains small amounts of water, because of its hygroscopic property, and when left exposed to the air will absorb more than half of its weight of moisture.

It boils at 290°C.

The Absorption of Glycerol

GLYCEROL is readily absorbed from the gastro-intestinal tract. Höber and Höber,[23] investigating the rate of absorption of equimolecular solutions of polyhydric alcohols from isolated loops of rat intestine, found that their absorption decreased with increase in their molecular weight. Approximately 73% of glycerol was absorbed as compared to 27% of erythritol and none of the mannitol. Johnson, Carlson and Johnson[29] found that 50% of the glycerol introduced into the intestine of anesthetized dogs, was absorbed during the first two to three hours. There was little absorption from the stomach.

Glycerol, like the hexoses, apparently unites with phosphates during its absorption from the intestinal tract. This is indicated by the increased content of acid-soluble organic phosphates in the mucosa of the intestine after oral administration of this compound (Laszt and Süllmann[35]).

A mixture of glycerol and phosphates, or even better, glycero-phosphate, administered together with glycocholic or taurocholic acids increases the rate and extent of absorption of a fatty acid such as oleic acid. On the basis of these observations Verzár and Laszt[83] concluded that the esterification of glycerol with phosphoric acid occurs in the mucous membrane.

Glycerol is absorbed rapidly if administered intraperitoneally, but slowly if given subcutaneously. The subcutaneous injection of small amounts usually presents no difficulties in animals, even though serious effects may be produced with larger doses. The intravenous injection of undiluted glycerol may result in occlusion of blood vessels.[10] Maignon and Grandclaude[47] reported the production of sclerosis of the veins in dogs by the injection of 3-5 cc. of pure glycerol.

The absorption of aqueous glycerol solutions from the blood stream starts immediately after injection.[78,37]

When applied to the unbroken skin, pure glycerol apparently is not appreciably absorbed.[10]

Acute Toxic Effects Induced by Glycerol

THE administration of glycerol to the horse, dog, cat, rabbit, rat and mouse in a single dose sufficient to cause death produced either restlessness or diminished activity, followed by an increased pulse rate, vomiting (in the horse and dog), occasional biting movements, a mild cyanosis, tremor, diuresis, some loss of equilibrium, severe clonic convulsions and death in coma.[31,40,69,58,29,68,55,36,10] Schübel[69] noted that the convulsions could be initiated by noise, an observation which we have made in the case of rabbits.[10]

Rabbits and rats show a marked inactivity, diuresis, loss of appetite and sometimes convulsions[10] in response to sublethal doses of glycerol. Kobert,[31] in 1906, called attention to the narcotic action of glycerol.

The Effect of Glycerol on Blood Pressure and Respiration

SCHUBEL[64] injected urethanized cats with doses of 1 cc/kg and noted an immediate drop in blood pressure of from 30 to 40 mm. The pressure rose again quickly, however, passing the normal, and remained elevated (40 mm. above normal) for 20 minutes. He attributed this temporary increase in pressure to an hydremic plethora, the water of the tissues being taken up by the circulation. Johnson, Carlson and Johnson[29] noted a transient fall in the arterial pressure, even after a small intravenous dose of glycerol. In addition to this, Trabucchi[78] observed an increased rate and magnitude of respiration in rabbits, which persisted after vagotomy. In our experiments on rabbits lethal oral doses produced a slowly failing circulation, lethal intraperitoneal injections produced a marked stimulation of respiration followed by a sudden drop of arterial blood pressure. Death was due to the combined effects of failing circulation and respiration.[10]

The Effect on Muscle, Intestine, Uterus and Blood Vessels

FROG muscles injected with or suspended in very dilute aqueous solutions of glycerol show a greatly increased and prolonged state of irritability, according to the observations of Santesson[66] in 1903, and Verzá and Peter[82] in 1925. Reach[61] found that glycerol caused a relaxation of the sphincter of the gall-bladder, when applied to the upper part of the duodenum. Pal and Prasad[52] noted an increase in the force and amplitude of intestinal contractions. No change in bowel activity was observed by Johnson, Carlson and Johnson in rats and dogs but there was a slight tendency toward softer stools in the human beings who in-

TABLE
LETHAL AND SUBLETHAL CONCENTRATIONS OF GLYCEROL (AS TAKEN FROM THE LITERATURE)

Species	Mode of Administration	Dose	Fate	Investigator
Child 2½ yr.	oral	300 gm	loss of consciousness, with recovery (stomach was pumped out)	reported by Kobert[31]
Child	?	?	fatal	" " " "
10 men and 4 women	oral	30 cc 3 times daily	no harmful effects	Johnson, Carlson and Johnson[29]
Horse	subcutaneous	300 cc	died in 27 hr.	Plosz[58]
"	"	100 cc	slight fever	" "
Dog	"	8-10 gm/kg	died in 24 hr.	Dujardin-Beaumetz and Audige, cited by Arnschink[2]
"	"	14 gm/kg	died in 3-4 hr.	Dujardin-Beaumetz and Audige, cited by Arnschink[2]
"	oral	up to 7 gm/kg	no illness	Lewin, cited by Arnschnik[2]
"	"	" " 8 " "	" "	Tschirwinsky, cited by Arnschnik[2]
"	"	7-11 gm/kg	intest. disturbances, vomiting	Arnschink[2]
"	"	35 % glycerol in diet	no ill effects	Johnson, Carlson and Johnson[29]
Rabbit	cutaneous	12 doses of 4.5 cc/kg	no signs of illness	Deichmann[10]
"	intraperitoneal	7 cc/kg	smallest dose lethal	" "
"	Subcutaneous	10 cc/kg	" " "	" "
"	oral	14 cc/kg	" " "	" "
Rat	intraperitoneal	5-6 cc/kg	killed from 40-80 %	" "
"	subcutaneous	4-5 cc/kg	may be fatal	Schubel[69]
"	"	8 cc/kg	minimal lethal dose	Pfeiffer and Arnove[55]
"	"	15 to 16 cc/kg	killed from 60-70 %	Deichmann[10]
"	"	13 cc/kg	killed 80 % (starved for 3 days)	" "
"	oral	5.7-14.8 cc/kg	no ill effects (starved 24 hr.)	Johnson, Carlson and Johnson[29]
"	"	41 % glycerol in diet fed 40 wk.	no ill effects . . .	" " " " "
"	"	21-23 cc/kg	killed from 40-60 %	Deichmann[10]
Mouse	subcutaneous	9-11 cc/kg	fatal to 80 %	Leake and Corbitt[36]

gested 90 cc. daily. Kobert[31] and Lewin[40] reported loose stools in human beings who ingested large amounts of glycerol. Schübel[69] found that glycerol had little or no effect upon the intestine and the uterus. This investigator also reported that a concentration of glycerol from 1:10 to 1:1000 had no effect upon the blood vessels and that it took a concentration of from 1:50 to 1:100 to exert a depressant action upon the isolated frog heart.

Effect of Glycerol on the Blood Picture

SIMON[72] observed anemia in rabbits injected intravenously. There was however a marked increase in the total leucocyte count count, a slight decrease in lymphocytes and a marked increase in the polymorphonuclear neutrophils. The injection of glycerol into the heart of rabbits was followed, in our experiments,[10] by a decrease in erythrocytes and in the hemoglobin concentration and an increase in leucocytes, findings which confirm Simon's observations.

Sugimoto[75] reported anemia in rabbits treated subcutaneously with 3 cc/kg. A pronounced decrease in erythrocytes and leucocytes and in the hemoglobin concentration was noted in our experimental animals[10] after the subcutaneous injection of lethal doses of glycerol. The repeated subcutaneous injection of sublethal doses had the same effect on the erythrocytes and the hemoglobin while the number of leucocytes fluctuated within a wide range.

Tompkins[77] obtained a well defined increase in the leucocytes of guinea pigs after intraperitoneal administration of glycerol in doses as great as those involved in injections of old tuberculin. There was a slight increase in monocytes and a marked increase in neutrophils. We[10] noted no significant change in the number of erythrocytes or in the hemoglobin concentration of the blood of rabbits during the first four to six hours after intraperitoneal injections of sublethal doses, but the number of leucocytes was increased. Twenty-four hours after the injection the hemoglobin was found to be below the pre-experimental level in some instances, while the leucocytes had returned to normal limits.

Johnson and Carlson[28] noted no change in the blood picture of human subjects who ingested 90 cc. of glycerol daily for 50 days. These results are in accord with our findings in rabbits to which glycerol was administered orally.[10]

Repeated applications of glycerol to the skin of rabbits induced no illness and no changes in the blood.[10]

Hemoglobinuria Due to Glycerol

LUCHSINGER[43] appears to have been the first to observe the production of hemoglobinuria in rabbits by subcutaneous injection of glycerol. His observations have been confirmed in the case of rabbits, dogs and rats, by Ustimowitsch,[80] by Miura,[49] by Johnson and Carlson,[28] by Simon,[70] by Sugimoto,[75] and by Deichmann.[10]

In 1937, Pfeiffer and Arnove[55] observed that 0.75 cc/kg was the smallest subcutaneous dose capable of producing hemoglobinuria in rats. They also found that the preliminary administration of ascorbic acid raised the parenteral dosage necessary to produce hemoglobinuria by 100% or more. The occasional production of hemolgobinuria in rabbits, but not in rats, by intraperitoneal administration was noted by Simon[70] and has been observed in our experiments.[10]

According to Simon,[72] the intravenous administration of glycerol to rabbits never produces hemoglobinuria, but our observations were not in complete accord with this statement. Of five rabbits, each of which received two injections of from 2 to 3 cc/kg into the heart, one showed a marked hemoglobinuria.[10] Kobert[31] reported the production of hemolysis by intravenous injections.

Miura[49] administered glycerol orally in sublethal concentrations to dogs and rabbits and observed no hemoglobinuria. These findings were confirmed by Johnson and Carlson[28] in the case of dogs, rats and rabbits, and by our observations on rabbits and rats.[10] Johnson, Carlson and Johnson[29] also failed to obtain hemoglobinuria in 14 human subjects who ingested 90 cc. of glycerol daily for 50 days.

Hemolysis Produced in Vitro by Gylcerol

AQUEOUS solutions of glycerol will hemolyze erythrocytes; this hemolysis appears to be more pronounced when the concentration of glycerol is low.

Jacobs, Glassman and Parpart[27] studied the rate of hemolysis by adding one part of blood to 500 parts of an isosmotic glycerol solution. They observed rapid hemolysis of the erythrocytes in man, rat, mouse, rabbit and guinea pig, and found that this could be retarded by increasing the CO_2 tension of the solution. The erythrocytes of the ox, sheep, horse, pig, dog and cat hemolyzed slowly, but this hemolysis could not be retarded by increasing the CO_2. They write: "By saturating an isosmotic gylcerol solution with alveolar air (though much lower tensions of CO_2 are effective) the time of hemolysis of red blood cells by glycerol may be prolonged to 50 or more times their normal values, this effect being readily reversible by the removal of CO_2 or by addition of $NaHCO_3$ to the solution. A striking inhibition of the effect of glycerol on the erythrocytes may be produced by traces of copper or of copper salts in the water (Jacobs and Carson[26]). This effect, which is not obtained with the blood of some animals, (cat, dog, sheep, ox), and only slightly with that of others, (rabbit, guinea pig), may be so pronounced with the blood of man, for example, that the use of water distilled in an ordinary copper still may change the time of hemolysis in 0.3 M glycerol at 20°C. from 30 seconds to 15 minutes or more. Equally striking effects may be obtained with the blood of the rat."

Schübel[69] studied washed erythrocytes placed in saline solutions and in water containing varying concentrations of glycerol. He noted no hemolysis in saline solutions containing up to 40% glycerol, while in 50% solutions hemolysis began after from 16 to 20 hours. As for aqueous solutions, a 20% solution of glycerol caused hemolysis

after approximately 15 minutes, a 30% solution after 30 minutes, and a 40-50% solution after 90 minutes. Calculated on an osmotic basis, a 2.55% solution of glycerol should be equivalent to a 0.9% solution of sodium chloride; however, glycerol was not found capable of replacing NaCl in isotonic solutions.

The Effect of Glycerol on the Body Temperature

IN 1887 Plosz[58] and Arnschink[2] reported that glycerol caused an elevation of the body temperature, an observation that was confirmed by Schellenberg,[68] who gave his patients a mixture of iodoform and glycerol. Johnson, Carlson and Johnson[29] observed no increase in temperature in a series of dogs to which glycerol was administered. In our experiments[10] rats, injected subcutaneously, suffered a drop in body temperature in direct relation of the size of the dose, but reacted variably to intraperitoneal injections in that no change in temperature occurred in some instances while in others the temperatures were depressed or elevated. Rabbits injected subcutaneously, orally or intraperitoneally, either maintained a normal temperature or developed a distinct fever.[10]

The Diuretic Action of Glycerol

IN 1876 Ustimoetisch[80] noted a diuretic action exerted by glycerol in dogs and rabbits. He observed also that the volume of urine secreted was always much greater after oral administration than after subcutaneous injection of glycerol. Dux and Löw[15] reported that the injection of this compound into the muscle of the frog was followed by loss of water which in some cases amounted to as much as one-fifth of the normal weight of the muscle. Schübel[69] produced a tenfold diuresis by the intravenous injection of glycerol into urethanized cats. The relationship between the degree of the diuresis and the concentration of the glycerol was studied by Cugusi[9] in 1922. He found that an intravenous injection of 1.2% gm/kg glycerol in a normal NaCl solution produced a notable diuresis; doses of from 2 to 2.4 gm/kg brought about a pronounced diuresis, while doses ranging from 4 to 6 gm/kg caused an inhibition of urine formation. Johnson, Carlson and Johnson[29] noted this inhibition also, but only when the intravenous dose of glycerol produced a marked lowering of the blood pressure at the same time. These authors noted no diuresis nor change in specific gravity of the urine in men and women who ingested about 100 gm. daily for 50 days, but rats and dogs on a diet containing 41 and 35% glycerol secreted approximately five times as much urine as control animals. Three of the five subjects of Lewis and Corley[41] who received 59 gm. of glycerol showed no diuresis.

The observations indicate that diuresis occurs after a moderately large dose of glycerol, but ceases if the toxic effects of this compound get the upper hand.

The Effect of Glycerol on Enzymes

A DOSE of 10 gm. glycerol per kilogram of body weight introduced into the stomach and upper portion of the intestine of an etherized dog, in Burge's experiments,[3] caused a 50% increase in the catalase concentration in the blood of the liver and the portal and jugular veins.

According to Allesandrini,[1] glycerol has no injurious effect on amylase, while Di Frisco[12] reported that pancreatic juice heated in the presence of glycerol does not lose its activity completely. Farber and Wynne[16] in studying the breaking-down of caseinogen by pancreatic protease in vitro found that glycerol "inhibits the initial rate of hydrolysis."

In our experiments subcutaneous injection of lethal concentrations of glycerol into rabbits and rats caused pronounced edema and hemorrhage at and near the site of injection. Within from 20 to 40 minutes after an intraperitoneal injection of 2 cc. of glycerol into rats, we observed accumulation of fluid in the peritoneal cavity which in some rats amounted to 10 cc. The oral administration of this compound was usually followed by an acute irritation of the whole intestinal tract. Rigor mortis usually developed in from three to five minutes after death.[10]

The Formation of Glycose from Glycerol

CREMER'S investigations[8] offered the earliest evidence of the transformation of glycerol into dextrose, though Ustimowitsch[80] and Plosz[58] had previously reported the occurrence of a Cu-reducing substance in dog's urine after the ingestion of glycerol. Cremer found that about 40% of the glycerol ingested by a phlorhizinized dog was excreted in the urine as "extra glucose." Chambers and Deuel[6] repeated Cremer's experiments and confirmed his observations. In 1904 Lüthje[44] administered glycerol to pancreatectomized dogs and found that this resulted in glycosuria. Voegtlin, Thompson and Duns[84] administered 20% aqueous solutions of glycerol orally and intraperitoneally to fasting rabbits and produced extensive and prolonged hyperglycemia. McLean[46] obtained substantiating data. Koike[32] and Yagi[89] introduced glycerol intraperitoneally into rabbits and noted hyperglycemia and an increase of lactic acid in the blood. The lactic acid so produced remained at a higher level than that resulting from the injection of equivalent doses of galactose, maltose, glucose, fructose and mannose. The hyperglycemic response was not attributable to the mobilization of liver glycogen but to the formation of glucose from glycerol. The excreted sugar could not possibly have been formed from the small amount of protein ingested. Complete conversion of glycerol into glucose may occur but the individual response is variable, perhaps because of incomplete absorption of the compound.

According to Ferber and Rabinowitsch,[17] when glycerol was administered on an empty stomach to healthy persons and patients in amounts up to 100 grams (diluted with water to 250 cc.), it produced hyperglycemia, the degree of which was directly related to the disturbance of the carbohydrate metabolism. In advanced cases of diabetes glycerol produced glycosuria. Parnas and Wagner[23] reported that the feeding of glycerol was responsi-

ble for an increased blood sugar concentration in a girl of nine who was suffering from a liver tumor. These reports indicate definitely that glycerol is a precursor of sugar in the organism, even though Kneip[33] does not share this opinion.

The Formation of Glycogen From Glycerol

CATRON and Lewis,[5] in observations on fasting rats over periods up to six hours, found that the glycogen content of the liver increased progressively to an average maximum of 143 mg. glycogen per 100 gm. body weight, in response to the administration of glycerol by means of a stomach tube. The amounts of liver glycogen formed during the two- and three-hour periods were similar to those observed after the absorption of glucose. Lederer[37] confirmed these observations in 1936. He found that the ingestion of glycerol by a fasting rat as well as the intravenous injection of glycerol into rabbits and dogs increased the hepatic glycogen. Reid[63] slowly infused glycerol into the veins of anesthetized cats and observed that liver glycogen was formed. Reid also found that glycogen deposition or breakdown in starving cats was in direct proportion to the body temperature, when other factors were kept constant. At 37° c. there was practically no change, at 38° c. there was a constant slight decrease, while at 40° c. a rapid decrease in the liver glycogen occurred. By means of perfusion experiments Lederer[37] brought proof that even in vitro, rabbit and dog livers formed glycogen from glycerol and that none of this power was lost in the icterus produced by experimental phosphorus poisoning.

These reports fully confirm the early observations of Weiss,[87] of Luchsinger,[43] of Salomon,[65] and of Hammersten,[19] who observed an increase in the glycogen of the liver after the feeding of glycerol to chickens and rabbits. Investigators who failed to make these observations either worked with amounts of glycerol which were too small, or carried out experiments of too short duration.[56,13]

The Effect of Glycerol in Counteracting the Action of Insulin and the Formation of Acetone Bodies

GLYCEROL leads to the formation of glucose and glycogen and therefore counteracts the effect of insulin and exerts a preventive action on the formation of acetone bodies. Noble and Macleod[51] recognized the fact that if glycerol is to exert any curative action on insulin coma it must be administered in a promptly available form. Pure glycerol proved to have little effect. The intraperitoneal or intravenous injection of a 20% aqueous solution of glycerol by Voegtlin, Dunn and Thompson[85] quickly banished the coma and convulsions induced in rats by a lethal dose of insulin. Reeves and Hewitt[62] also noted that the administration of glycerol was of assistance in combating the phenomena of hypoglycemia induced by insulin. Trabucchi[78] injected glycerol in the form of a 30% solution into rabbits, using a dose of 5 gm/kg, and followed it immediately by an intramuscular injection of insulin. He noted that the glycerol disappeared more rapidly from the circulation blood of these animals than from that of non-insulinized rabbits. Decrease in the formation and excretion of acetone bodies in diabetics and normal fasting persons and animals following the ingestion of glycerol was reported by Hirschfeld in 1895,[22] by Satta in 1905,[67] by Lang in 1915,[34] and by Chambers and Deuel in 1925.[6]

The Protein-Sparing Action of Glycerol

LITTLE definite information is available regarding the possible protein-sparing action of ingested glycerol. Dubois and Combemale[14] studied the urea excretion before, during and after glycerol feeding; in their experiments the reduction in the excretion of urea was too small to warrant the assumption of a conservation of the proteins. Very similar results were obtained by Munk,[50] Lewin,[39] and Tschirwinsky,[79] Strack,[74] however, observed a definite protein-sparing action and Chambers and Deuel[6] found the protein-sparing activity of glycerol to be similar to that of the monosaccharides.

Glycerol apparently will exert a protein-sparing action only when it is administered in high but tolerable concentrations.

The Effect of Glycerol on the Respiratory Quotient

THE effect of glycerol on the respiratory quotient is controversial. McCann and Hannon[45] and Johnson, Carlson and Johnson[29] found no consistent response on the part of the respiratory quotient to the ingestion of from 40 to 100 gm of glycerol by diabetic patients or normal persons. On the other hand, Chambers and Deuel[6] found a reduction of the respiratory quotient in three phlorhizinized dogs, after the oral administration of 8.5 gm. of glycerol. They explain their results by saying: "The decrease in R.Q. from 0.703 to 0.678 after giving glycerol indicates that there was no general oxidation of the ingested glycerol, which has an R.Q. of 0.857, nor of the glucose into which it was converted. The reduction in the R.Q. from 0.703 to 0.678 may be accounted for as due to the oxidation of the 2 atoms of hydrogen liberated in the reaction: glycerol = ½ glucose."

Glycerol as a Substitute for Carbohydrates in the Diet

IN 1933, Johnson, Carlson and Johnson[29] kept rats for 40 weeks on a diet consisting of 20% starch, 41% glycerol, 18% casein, 10% butter, 5% yeast, 4% inorganic salt mixture and 2% agar. The animals grew as well as control animals given a diet containing 61% starch. Three dogs kept for 36 weeks on a diet containing 35% glycerol, 17% carbohydrates, 24.5% protein, 7% fat, 2.5% yeast and 14% bone meal also showed the same gain in weight as control dogs kept on a diet in which none of the carbohydrate was replaced by glycerol. By reducing the food intake of these dogs while following their weight changes these workers demonstrated that glycerol is not calorically equivalent to glucose when it makes up more than 35% of the diet. They also studied 10 men and four women ingested 30 cc. of 95% glycerol with orange juice three times daily after each meal for 50 days. These subjects developed some tendency

towards increase in body weight, and described such subjective effects as: "increased thirst occasionally (two subjects), and occasional sensation of warmth in the stomach after ingestion, especially in those instances when the glycerol was taken not with a meal but on an empty stomach (four subjects), and even a feeling of particular well being and exceptional freedom from colds (one subject). There were no reports of sleeplessness, talkativeness, or excitement." In 1929 Ferber and Rabinowitsch[17] gave oral doses of 100 gm. of glycerol in 250 to 300 cc. of water to 60 patients and noted little if any effect therefrom. No instance of gastro-intestinal disturbance occurred and only five complained of any discomfort, the latter consisting usually in a throbbing headache or dizziness, which these authors feel may well have been due to lack of food since the glycerol was taken on an empty stomach, 10 hours after the last meal. Flury and Zangger in 1928[18] stated that oral doses of 100 gm. of glycerol produced vomiting and headache in humans, but the more recent observations indicate that this holds true only in isolated cases. In 1937 Holck[25] confirmed the harmlessness of a diet containing 20% glycerol, on which rats were kept for 11 weeks. According to Delaunary and Accoyer's investigations,[11] reported in 1936, the normal rat is capable of assimilating a maximum of 0.4 gm. glycerol per 100 gm. body weight per day when the glycerol is mixed with the food.

The Effect of Glycerol on Uric Acid Excretion

ROSENFELD[61] and Quick[60] among others have called attention to an increased elimination of uric acid concretions and sediment after glycerol ingestion. Hansen and Kamm[20] offer the following explanation for the beneficial influence of glycerol on nephrolithiasis: by dissolving out the inorganic matrix of the uric-acid calculi, glycerol breaks them up into fine granules which settle out slowly and therefore remain suspended in a mixture of urine and glycerol instead of adhering to the wall of the bladder; in this free state they are easily voided with the urine. Johnson, Carlson and Johnson[29] found no significant change in the excretion of uric acid in their human subjects after the latter had ingested 90 cc. of glycerol daily for 50 days. Their subjects were healthy young people and consequently these findings would have little bearing on the question of the disintegration and elimination of pre-existing uric-acid calculi.

The Effect of Glycerol on Reproduction

THE normal reproduction of rats is not influenced by the substitution of glycerol for starch up to a point where the former comprises 41% of the total food intake. Pregnancies did not occur when rats received 61% of their diet in the form of glycerol.[29]

The Excretion of Glycerol

THE rate of the urinary excretion of glycerol depends upon a variety of factors such as the rate and mode of its introduction into the body, the composition of the diet, and the condiiton of the excretory organs. Even prior to 1887, the excretion of glycerol in the urine of human beings and dogs had been observed by Tschirwinsky,[79] Catillon,[4] and Arnschink.[2] According to Delaunay and Accoyer,[11] rats kept over a prolonged period on a diet including glycerol excrete all the glycerol absorbed in excess of 4 gm/kg.

Glycerol Absorption in Tuberculosis and Certain Other Pathological Conditions

THE filtrate employed in the treatment of tuberculosis is prepared from a concentrated glycerine-broth culture of human tubercle bacilli and is so titrated that it contains 50% glycerol and 1 mgm of tuberculin to each cubic centimeter of solution. At times glycerol bouillon gives a positive skin reaction when administered simultaneously with tuberculin to tuberculous guinea pigs. Heise and Brown[21] noted a positive reaction from glycerol bouillon and tuberculin in 4% of 443 cases of tuberculosis in adult human beings. Höchst[24] observed a positive reaction to injections of concentrated glycerol bouillon in 83 of 93 tuberculin-positive children and in 16 of 17 tuberculin-positive adults. Of 112 tuberculin-negative children, under the care of Keller and Dölter,[30] none reacted.

Long and Vorwald[42] administered glycerol, both orally and subcutaneously, to tuberculous rats over long periods. They observed an enhanced growth of tubercule bacilli in the lesions and a general increase in the severity of the disease. The majority of tuberculosus rats fed upon an excess of palmitic acid had smaller lesions than did tuberculous rats on a control diet.

Woolf[88] has reported good results in the treatment of hemorrhoids with a mixture of 40% glycerol, 20% phenol and 40% witch hazel, in conjunction with a 5% solution of phenol in almond oil.

Acording to Compton,[7] cancer cells of the epithelial type, in vitro, possess a high degree of resistance to the toxic action of pure glycerol.

Glycerol inhibits the action of staphylococcic toxin in causing necrosis of the skin of guinea pigs, an inhibition according to Smith[73] is specific for this toxin.

In 1931, Tashiro and Oliver[76] studied the possible antagonistic action of glycerol and cholesterol against bile salts. They found that neither of these compounds prevented erosion of the gastric muscosa, or death, in rabbits and guinea pigs injected intravenously and intraperitoneally with toxic doses of bile salts.

That glycerol prolonged the life of suprarenalectomized cats was demonstrated by Marine and Baumann[48] in 1927. Glucose appeared to be about equally effective in this respect.

Pathological Changes Caused by Glycerol

THE subcutaneous injection of glycerol into the horse or dog, produced edema at the site of injection, inflammation of the lungs and intestinal wall, and hyperemia of the liver and kidney.[58]

After injecting three rabbits subcutaneously with doses of glycerol sufficient to kill them in 24,

48 and 92 hours, respectively, Simon[70] found lesions mainly in the subcutaneous tissues, the liver and the kidney. At the site of the injection the glycerol had destroyed the framework of the connective tissue, the adjacent fascia had disintegrated into longitudinal fibrils and the muscle fibers had lost their cross-striations. The liver showed an increase in connective tissue, atrophy of the hepatic cells, parenchymal hemorrhage, perivascular leucocytic infiltration and cellular atrophy in the periphery of the lobules. The glomeruli and the tubules of the kidney were also involved in an inflammatory process. Piras[67] injected subcutaneously daily doses of glycerol ranging from 0.5 to 3 cc. per rabbit for a period of 45 days, killed the animals by bleeding, and studied the histopathological changes in the tissues. The organs primarily affected were the liver, kidneys, intestine and lungs. The liver and kidneys showed marked hyperemia, dilatation of the vessels, hemorrhage, and cloudy swelling. Pulmonary lesions were common to all rabbits, the degree of injury being proportional to the dose injected. There were signs of extensive injury of the mucosa of the intestine, with inflammation, congestion and hemorrhage. According to Tompkins,[77] the repeated injection of sublethal doses of glycerol into guinea pigs caused no permanent pathological changes. Johnson, Carlson and Johnson[29] observed no gross or microscopic tissue changes in rats fed glycerol for a prolonged period. Kobert,[31] however, reported in 1906 that the repeated ingestion of large doses of glycerol leads to a chronic inflammation of the stomach and intestines.

Summary

GLYCEROL is absorbed from the peritoneum, gastro-intestinal tract and subcutaneous tissues; its penetration of the intact skin appears to be negligible.

Toxic doses of glycerol produce a train of characteristic signs and symptoms, and when administered in sufficient quantities, death. The quantity necessary to produce death varies with the mode of administration, being least if given intraperitoneally, intermediate if injected subcutaneously, and largest if given orally. (The lethal dosage by the intravenous route is obscured by the secondary effects of embolism and other direct effects upon the blood). It is not possible to kill rabbits or rats by cutaneous application. Partial starvation decreases the resistance of animals to glycerol poisoning.

The intravenous injection of even small doses is followed by a transient fall in the arterial blood pressure and by an increased rate and magnitude of respiration. Lethal oral doses produce a slowly failing circulation, while lethal intraperitoneal injections produce a marked stimulation of respiration, and finally a sudden drop of the arterial blood pressure. Death is due to the combined effects of failing circulation and respiration..

Glycerol increases the degree of irritability of muscle, causes relaxation of the sphincter of the gall bladder, and appears to increase the force and amplitude of intestinal contractions.

It has little or no effect upon the uterus.

The portal of entry, as well as the dose of glycerol, determines the specific effect on the blood picture. Aside from a temporary concentration of the blood, due to the loss of water, the general picture is as follows. Subcutaneous injection produces a decrease in the number of erythrocytes and in the hemoglobin concentration, together with hemoglobinuria. The number of leucocytes fluctuates above and below the normal. Intraperitoneal administration is followed by an increase in the number of leucocytes and by insignificant changes in the number of erythrocytes and in the hemoglobin concentration. Hemoglobinuria may develop in isolated cases. No significant blood change has been reported after oral administration or after cutaneous treatment but intravenous injections are followed by decrease in the erythrocyte count and in the hemoglobin concentration and an increase in leucocytes, after a temporary increase in all the formed elements by reason of the direct effect of glycerol in concentrating the blood. Hemoglobinuria may be produced in isolated cases treated intravenously.

The rate of hemolysis of erythrocytes when whole blood is added to isosmotic glycerol solutions in vitro varies widely for different species of animals. Washed red blood cells are very resistant to hemolysis if placed in a saline-glycerol solution, but they are hemolyzed readily if placed in an aqueous glycerol solution, the rate of hemolysis varying inversely with the glycerol concentration.

The effect of lethal and sublethal doses of glycerol on the body temperature does not appear to be uniform for all species or for different modes of administration. Both pronounced fever and subnormal temperatures have been observed.

Diuresis appears to be more pronounced after oral than after subcutaneous administration, but it is independent of the mode of entry of glycerol into the body. Toxic concentrations in the body, however, inhibit the formation of urine.

A portion of the absorbed glycerol is transformed in the organism into glucose, and thence into glycogen. Glycerol, therefore, is antagonistic to the action of insulin and to the formation of the acetone bodies. Various investigators have employed glycerol as a partial substitute for carbohydrates in the diet with apparently satisfactory results. The total replacement of carbohydrates by glycerol prevents pregnancy.

Glycerol appears to have a protein-sparing action if administered in concentrations well below the toxic level.

The reported effect of this compound in decreasing the respiratory quotient needs verification.

Ingestion of glycerol appears to favor the excretion of uric acid. The mechanism involved is not fully understood.

Glycerol may be excreted as such in the urine if sufficiently large single doses are administered, and also if small amounts are given over a prolonged period.

Glycosuria is not common after absorption of glycerol.

The pathological changes which have been reported in cases of experimental glycerol poisoning are those of acute toxic damage to the liver, kidney and lungs. The gastrointestinal tract may suffer after the administration of large oral doses and the subcutaneous tissues may reveal pronounced local damage at the site of injection.

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Toxic Limits

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THE foundation of modern science and modern technique is the process of weighing, measuring and counting. The step from the empirical state to modern science, from skilled handiwork to mechanical performance was possible only after methods for specific weighing and measuring had been found. Exact examinations of air and its contaminants in factories were great rarities even 20 and 15 years ago; indeed determinations of dust concentrations were the only such measurements made to any appreciable extent and these were done in English speaking countries alone. No other instruments were available for investigations in factories except those for determining the dust contents. (In the United States, the sugar-tube, Palmer apparatus, Greenburg-Smith impinger; in England, Owens dust counter; in South Africa, Kotzé konimeter.) The methods and instruments used in laboratories have also been used in factories, instruments difficult to handle and transport, often not accurate. All this is different today.

We now have various instruments suitable for use in factories, although the truly exact instrument and the precise method is still lacking for certain purposes. On the contrary we even suffer from too many offers of instruments made by firms and scientists. Their only small deviation from already existing instruments makes it more difficult to compare the results, since the new instruments often do not guarantee any greater accuracy.

R. R. Sayers[1] published in 1927 data about the most important poisons, briefly summarizing them. As far as I know, Zangger[2] in 1928 was the first author to compile a table "about the effect of irritant and poisonous gases and vapors." For many gases he specified the exact concentration in milligrams per liter which would kill immediately or be fatal after ½-1 hour. He defined which concentration may be borne with large, small or no damaging effect for a period of time. The compilation is supported for the largest part by animal experiments, made by K. B. Lehmann and his collaborators, Zangger, U. S. Bureau of Mines, Haldane, Henderson and Haggard. Experiments on human beings (experiments on the author himself) are rarely referred to. An extension and completion of this table may be found in the book, Noxious Gases by Flury and Zernik.[3]

The table, "Maximum Allowable Concentrations," compiled by Bowditch[4,5] was especially meritorious as it included tabulation of regulations of other governmental authorities in addition to those suggested for Massachusetts. These had been referred for criticism to more than a score of the leading authorities in this country and abroad prior to their promulgation. This table has been revised and extended by himself, C. K. Drinker, P. Drinker, H. H. Haggard and A. Hamilton[6]. By formulating the problem into *"allowable concentrations"* it has been taken out of the field of theoretical research and *transferred to the practical industrial hygiene.* But the ready availability of apparatus easy to handle and of tables of toxic limits established by well known scientists to which the results can be referred for interpretation may lead to the undesirable situation that men not well trained in industrial hygiene will underestimate the difficulties of obtaining truly representative results. In addition there is the danger that they will employ these results beyond their limits of usefulness and so draw unjustifiable conclusions.

Therefore the *value of the limits,* the *practical purpose* and the *limitations* of their use may be discussed in the following lines.

A SHARP distinction must be drawn between acute and chronic action which must be applied especially to those materials which produce acute and chronic occupational poisoning (benzol, toluene, xylene, carbon disulfide, trichlorethylene, aniline, ozone, etc.). The "allowable concentration" is much higher where there is danger of acute poisoning than under conditions where there is continuous inhalation for a long time. It is evident that it would be out of place to demand the same limiting value of contamination where the workers are only occasionally exposed to the danger of acute poisoning (transferring benzol, trichlorethylene, carbon bisulfide, etc., or similar intermittent work performed at extended intervals) as in places where there is danger of continuous inhalation for a period of weeks or months.

The Massachusetts publication quoted above seems to give the "allowable concentrations" for those actions which are most important in industrial hygiene: the chronic influence of benzol and its homologues, the acute poisoning by CO. Although the expert knows or is able to find out for which situation the given value is applicable, it would be expedient *to specify in the table the limits for acute and those for chronic poisonings.* If that is not possible or not necessary for some substances, it should be stated clearly whether the quoted value refers to acute or chronic action.

Of course, the conceptions of "acute" and "chronic" are not sharply differentiated. But generally we may call poisonings and damages acute when they can take place within a working day; and we may call them chronic when they need a longer time to develop — usually weeks or even years.

A specification of the concentration fatal in a half hour or less, although scientifically interesting, is of little value for practical industrial hygiene as pointed out below.

As examples of the damages which arise after harmful action of years or decades may be mentioned silicosis, bladder cancer of aniline workers, the symptoms of chronic alcoholism, certain damages by x-rays and radium, some serious forms of carbon bisulfide poisonings, mercury shakes, the changes of the vessels and as a rule the pareses as a result of lead poisoning.

But if a man working with lead for many years

falls ill with lead colic, i.e., the subacute form of lead poisoning, it is correct to suspect that he has recently absorbed an exceptionally large amount. The same cause — a larger amount of absorbed poison — may also be considered in case a man working with mercury for many years acquires gingivitis and diarrhea; and if serious blood change in a man working many years with benzol is not started by infectious disease, it is possible that it was caused by a larger recent absorption of benzol.

Many animal experiments are at hand concerning concentration and duration of air contaminations which cause temporary or lasting injury — but their application to man is either not feasible or to be done only very cautiously.

There are important differences between the different species of animals. The fatal concentration of carbon monoxide poisoning at 32° C. is above 0.12% by volume for the rat; above 0.25%, for the guinea pig and above 0.38% for the rabbit.

Flury and Zernik[3] tell us that cats, mice, rats are very sensitive to phosgene; dogs are less sensitive, rabbit still less. Horses are less sensitive than human beings. When the driver died in gas attacks during the World War, the horses sometimes showed only slight symptoms of poisoning.

Our conclusions therefore on acute poisonings have to be based preferably on the experiences we have had with man. For chronic poisoning and damages the animal experiments fail entirely to be useful for our purpose because it is impossible to determine the comparability of the time required for injury to develop in animal and in man.

A study of conditions which may produce chronic poisonings presents a number of difficulties. The contamination of the air can be determined during the whole working shift very well, but is it impossible to ascertain the circumstances under which the injurious material was absorbed during months and years gone by.

If it is admitted that the conditions affecting industrial health have improved in the course of years — which is true in general, but not in every single case — or that the working time is shortened, then the amount of injurious material absorbed in a working day was larger in former times than it is now. But it may also be that the damaging material is not used such a long time, as the case history may tell us, or that its concentration has been increased recently; such as unnoticed substitution of benzol for benzine or a benzine-benzol mixture, or substitution of lead for color containing little or no lead.

Therefore it must be remembered that all results are approximate values only. But the most important materials, especially in regard to their chronic poisonings (benzol, carbon disulfide, lead) have been studied with such thoroughness and skill by our best industrial hygienists that the obtained figures are actually practicable as limits.

But the observations about some other substances (cadmium, chlorine (chronic), dichlorethylene and many others) are too limited in number and not clear and reliable enough to make even partly safe conclusions. If figures about such substances are included in the list at all *a mark of interrogation has to be added.*

The limits are approximate, not only on account of the difficulty of most of the observations, but also on account of differences in sensitivity of individual persons and on account of different amounts of the contaminated air absorbed by the worker under different circumstances. For instance, the worker when working hard inhales much more of the contaminated air (up to 50-60 liters per minute) than when doing light work (8-10 liters per minute).

But these lists of toxic limits give the practical industrial hygienist essential data for his work; they save laborious studies of literature and give him the results obtained by those carrying on research and basic investigation. *Therefore we have to be thankful for the compilations of the limits by their authors.*

How are the "limits" to be utilized? For which purposes may they be used? What is their immediate practical advantage and when may they be applied?

FIRST of all: The limits strongly support the industrial hygienist in arriving at an opinion as to whether the exposure is or is not excessive, and, if so, in convincing incredulous employers and engineers (under certain circumstances) that the contaminated air in their rooms is injurious even though the contamination cannot be readily seen or smelled. The data on the exposures, if properly substantiated, act as impartial arbitrators between the man recommending control measures and the man persisting in the present situation.

The limits are useful in construction and testing exhaust systems. Naturally you will try to remove every contamination of the air, because only that gives complete security. If that is not possible, you must insist that the ventilation has at least such an effect as to reduce the exposure to less than the "allowable concentration" in the breathing zone. If that is impossible, you have to try to bring about hygienic conditions by changing the operation or by abandoning the use of the poisonous substance.

I might indicate that exhaust ventilation seems to be considered too much as the principal remedy nowadays, as the respirator was considered some years ago though with much less foundation. Other procedures mentioned above are sometimes forgotten today, although they have attained great success in industrial hygiene.

If the limits are used, it must be definitely ascertained that *the evaluation of the contamination in the air is reliable.*

Because the testing devices are reasonably priced, easy to handle and because the methods of examination in laboratories are frequently simplified, there is (as remarked previously) the danger of unqualified men using them. Therefore the following has to be stressed:

Serious acute poisonings, especially fatal ones, arise through unforeseen accidents — as leaking of vessels, pipe lines, or by unexpected formation of gases in larger amounts or by other abnormal

events. Also, by exceptional atmospheric conditions where the removal of gases is not effected by strong mechanically driven exhaust systems.

A locksmith was found dead beside his forge fire where he had been seen working 20 minutes before. As an exhaust pipe had been installed in this place and the man had worked here for many years, the technician thought a CO poisoning impossible—the blood of the dead man however was found to be saturated with CO. Abnormal cold and severe storms had interfered with the natural up-draft existing at other times. I have seen CO poisoning with unconsciousness in some working places of a large plant in presence of such abnormal weather; the workers had worked in these places without disturbance of health for months.

I have seen poisonings by arsine as a result of using too strong an acid so that the solution was not neutralized; as a result of too much zinc powder being added; and as a result of an exhaust pipe being obstructed by the formation of foam.

Such circumstances caused by abnormal accidental events cannot be investigated by air examination because we do not know all contributing factors and therefore we are not able to reproduce these circumstances (to say nothing of the danger to the investigator). Therefore, as mentioned above, data about the deadly concentration would be of little practical value.

In all other circumstances it is self evident that *only numerous samples* of air give a true picture of the existing conditions, if taken *at different times* and during different working processes and in the exact working place at the *breathing level* of the worker. Samples taken off-hand are without any value.

THE investigator has to be certain that the working processes during the time of examination are the same as at other times. The investigator can be misled intentionally or unintentionally. The occurrence of illness should of course induce the improvement of the working conditions. When we investigate we have to know if such changes have taken place or not. It is sometimes difficult to find that out.

Another difficulty which may be overcome only by patience is the fact that all persons concerned — even involuntarily — work more "according to prescription" if observers and supervisors are present.

In addition it must be taken into consideration that little variations — produced intentionally or not — are able to change the whole picture; small changes hardly detectable in the process produce considerable differences in the amount of contamination in the air; season (open or closed windows), and weather also influence the picture.

An electrolytic nickel plating bath delivers more hydrocyanic acid gas, a chrome plating bath more chromic acid mist, when the current is heavier or the treated objects larger. The amount of lead fumes depends, among other factors, on the temperature of the lead bath, the zinc oxide fumes on the temperature of the molten alloy. More vapor escapes in an apparatus for degreasing by trichlorethylene if the trichlorethylene is heated more and if it is not cooled before emptying. The quantity of escaping vapors changes in the usual degreasing apparatus with the temperature of the water used in the cooling coil, with the size of the objects and their number and with the frequency and speed with which they are taken out.

Ball mills pulverizing rock have often been submitted to my inspection and I was shown they were working without escape of dust, — it was soon after loading large rock. But half an hour later, when the material has been reduced to fine powder, much dust was escaping into the breathing zone.

To give some examples of numerical differences: Dust concentrations found in my investigations in grinding shops[7] in the summer by the Owens dust counter have averaged between 600 and 1500 particles per cm^3 in the same room, at the same speed of rotation of the sandstone wheels but grinding different kinds of knives. In winter, when the windows were closed, the figures ran up to 3700 at the breathing level of the worker. In the air of the room in general the dust figures averaged to 560 in summertime, 3300 in winter. Bowditch and Elkins[8] found in the coating room of an artificial leather factory on different days in March 130-200 parts per million of benzol; concentrations of 300-1300 ppm of naphtha vapor were detected during the work done by one worker in a factory using rubber cement. Greenburg and corroborators found in a rotogravure printing plant 50-1060 ppm of benzol in different parts of the press room.

I think it is not necessary to stress that all the difficulties mentioned above have to be regarded especially if there is a compensation case, or if there is a question of negligence.

Summarizing: A well-trained industrial hygienist only is able to make investigations in industrial hygiene and especially for answering the question, if, when or how often limits are transgressed; and besides he can answer only after a long-lasting and exact examination.

It should never be forgotten, that all our limits have their origin from health examination of workers and that only from comparison between the results of these physical examinations and the content of contamination in the air do the limits result. All the mentioned uncertainties and difficulties can be avoided in many cases if we refer to this origin. If there is a danger of chronic poisoning the thorough investigation and examination of the workers especially for early symptoms give us a sure answer in cases where the time is long enough for acquiring symptoms of chronic poisoning.

We may also see if the contamination is able to produce acute poisoning by examining the workers at the end of the daily working time.

A special consideration must be given to the question if and how far the results of air examinations and their relation to the limits are able to contribute in determining the occurrence of an *occupational disease*.

The question of whether there is an occupational disease presents itself as a medical question. A physician only is able to answer it after considering all the clinical symptoms and after a thorough medical examination executed with all necessary aids.

The clinical pictures (and the pathological one in the autopsy) of the more frequent occupational diseases (chronic poisonings by lead, mercury, benzol, silicosis, to some extent carbon disulfide, and certain acute poisonings as CO) have been studied so carefully and are so well established that the diagnosis can be made with greatest certainty — by a physician experienced in this field as is the case with every other medical diagnosis.

We have mentioned above the difficulties and uncertainties of air examinations. We cannot guarantee that the air contamination during the investigation was the same as in the time before the poisoning occurred. Therefore negative findings in air contaminations or findings below the limits are never able to shake such a positive medical diagnosis though under certain circumstances it is possible that a former occupation may have been responsible for the disease. Positive findings on the other hand are able to support it. Furthermore if we have a clinical picture that according to medical opinion does not correspond with the well known picture of this poisoning nor with signs of its rare varieties and therefore the physician denies an occupational disease, then the findings of air contamination above the limits is no proof of a causal connection between the illness and the contamination; the illness may have originated entirely independently of the contamination because of an internal or another external cause.

But there is another method which gives us an insight into the circumstances under which the ill man worked prior to his disability and which should be practiced in chronic poisonings if it can be done shortly after the beginning of his illness: The physical examination of the fellow-workers (still working, disabled and dismissed). If the symptoms of the same illness are found, it confirms our diagnosis of the case in question. Negative findings are of small value, because the sick man may have been more exposed to the damaging substance or may be more sensitive.

In acute poisonings the examination of the fellow-workers should be made at the end of the shift.

There is however, a long list of less explored substances (most at present rarely used), the clinical pictures of which are not clearly worked out as yet — nor are their toxic limits. We are able to conclude that there is a causal connection if certain substances are found in the air and contemporarily symptoms of illness determined in a worker and if these symptoms agree with those mentioned in the literature as a consequence of this poison or if similar pictures are to be seen among the fellow-workers.

But cases occur when a positive medical diagnosis alone is not sufficient. For compensation cases and for prophylaxis it may be of greatest importance to know through which kind of work the illness is acquired.

If the examination of the air fails to give exact proof, then all chemical and other processes should be subjected to the opinion of experienced experts in technology, chemistry and industrial hygiene. In such cases all the impurities possibly present in the substances used are to be considered.

I wrote above "experienced physicians." The universities (medical schools) should give better instructions to students of medicine in industrial hygiene and in occupational diseases. Especially in the latter field, there should be established short courses for physicians also. The legislation, the industrial commissions, the insurance companies, the employers and the employees should take advantage first of all of such trained physicians and thus give them occasion to acquire still greater experience.

Summary

TO DEFINE toxic limits is of the greatest scientific and of the greatest practical value.

2. It should be made evident whether the toxic limit applies to acute or chronic poisoning, if it is well founded or doubtful.

3. The limits are valuable help in showing the necessity of prophylactic measures in a plant for the construction of new and for testing of existing installations. But the examinations should be made very carefully and by experienced industrial hygienists. In determining the severity of exposures resulting from existing installations, the medical examination of workers often gives a better insight.

4. It is the field of medical diagnosis to ascertain the presence or absence of an occupational disease. This diagnosis made by an expert physician cannot be shaken by a contrary result of air examination, but if this result is certainly representative of the worker's exposure, it may point to the desirability of further investigations.

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National Conference

—of Governmental Industrial Hygienists—

THE complete gamut of industrial hygiene is covered in the papers and committee reports of the Third Annual Meeting of the National Conference of Governmental Industrial Hygienists held at Bethesda, Maryland, April 30, May 1, 2, 1940. The Transactions have been published and include, as a valuable entity in itself, the discussions on the subjects presented. Copies of these Transactions are available on application to Division of Industrial Hygiene, National Institute of Health, Bethesda, Maryland.

The titles of the technical papers and reports of committees are given below.

April 30, 1940—Morning Session

OPENING Remarks—DR. R. R. SAYERS, Director, United States Bureau of Mines.

Address of Welcome—DR. THOMAS PARRAN, Surgeon-General, United States Public Health Service.

April 30, 1940—Afternoon Session

SYMPOSIUM on Interdepartmental Relationships:
"The Viewpoint of a State Health Department" — LESTER A. ROUND, Director of Health, Rhode Island.
"The Viewpoint of a State Industrial Commission"—THURSTON A. WILSON, Chairman, North Carolina Industrial Commission.
"The Physician and the Industrial Hygiene Services"—DR. C. M. PETERSON, Secretary, Council on Industrial Health of the American Medical Association.
"Relations of Official Industrial Hygiene Services with Industry"—DR. C. D. SELBY, Medical Consultant, General Motors Corporation.

May 1, 1940—Morning Session

MEDICAL SECTION: "A Physical Examination Program for Industrial Workers"—DR. PAUL A. BREHM, Supervisor, Industrial Hygiene Unit, Wisconsin.
"The Lead Hazard in the Storage Battery Industry"—DR. CARL A. NAU, Director, Division of Industrial Hygiene, Texas.
"Industrial Health and the Industrial Medical Program" —DR. KENNETH E. MARKUSON, Director, Bureau of Industrial Hygiene, Michigan.
"Syphilis Case Finding and Case Holding in Industrial Workers"—DR. ALBERT E. RUSSELL, Surgeon in Charge, Syphilis Control in Industry, U. S. Public Health Service, Chicago.
NON-MEDICAL SECTION: "Applied Principles of Exhaust Ventilation"—WILLIAM N. WITHERIDGE, Ventilation Engineer, Bureau of Industrial Hygiene, Detroit, Michigan.
"Problems in Industrial Hygiene Air Sampling"—DR. W. G. FREDRICK, Chief Chemist, Bureau of Industrial Hygiene, Detroit, Michigan.
"Methods of Analysis of Air-Borne Contaminants"—ALFRED N. SETTERLIND, Chief Chemist, Division of Industrial Hygiene, Illinois.

May 1, 1940—Afternoon Session

INTRODUCTORY Remarks"—DR. P. A. NEAL, Acting Chief, Division of Industrial Hygiene, U. S. Public Health Service.
"Recent developments in Environmental Studies in Industry"—R. T. PAGE, Associate Public Health Engineer, U. S. Public Health Service.
"Remarks on the Research Laboratories of the Division of Industrial Hygiene"—DR. P. A. NEAL, Acting Chief, Division of Industrial Hygiene, U. S. Public Health Service.
"Recent Analytical Problems in Evaluating the Industrial Environment"—F. H. GOLDMAN, Associate Chemist, U. S. Public Health Service.
"Pneumoconiosis Due to Certain Silicates"—W. C. DREESSEN, Passed Assistant Surgeon, U. S. Public Health Service.
"Protective Ointment for the Prevention of Poison Ivy Dermatitis"—LOUIS SCHWARTZ, L. H. WARREN, and F. H. GOLDMAN, U. S. Public Health Service. (Presented by Dr. Schwartz).
"The Relative Toxicity of Lead and Some of Its Common Compounds"—LAWRENCE T. FAIRHALL, Principal Industrial Toxicologist, U. S. Public Health Service.
"Recent Studies and Theories on the Mechanism of the Action of Carbon Monoxide Poisoning"—W. F. VON OETTINGEN, Principal Industrial Toxicologist, U. S. Public Health Service.
"Fatigue and Hours of Service of Interstate Truck Drivers"—R. H. FLINN, Passed Assistant Surgeon, U. S. Public Health Service.

May 2, 1940—Morning Session

REPORT of Committee to Study Methods of Appraisal of Industrial Hygiene Problems in a Locality, 1939-1940"—DR. CRIT PHARRIS, Chairman.
"Report of Committee on Technical Standards"—THEODORE HATCH, Chairman.
"Report of Committee on Industrial Hygiene Education"—DR. CARL A. NAU, Chairman.
"Report of Committee to Study Methods for Securing Effective and Uniform Reporting of Occupational Diseases and Other Illnesses Among Workers"—DR. J. M. MCDONALD, Chairman.
"Report of Committee on Administrative Development of Industrial Hygiene Activities in the State Through Local Departments of Health—DR. K. F. MARKUSON, Chairman.
"Report of Committee on Administrative and Technical Relationships"—DR. ALBERT S. GRAY, Chairman.
"Proposed Recommendations for the Installation and Safe Operation of Dry Cleaning Systems Using Chlorinated Solvents"—
"Report of Committee on Minimum Qualifications for Industrial Hygiene Personnel" — J. J. BLOOMFIELD, Chairman.
"Report of Committee on Appraisal and Uniform Reporting of Industrial Hygiene Activities"—J. J. BLOOMFIELD, Chairman.

Data on Industrial Hygiene

—First Call for Papers for the Annual Meeting—

ORIGINAL research in the various phases of industrial hygiene and results of investigations of occupational disease exposures which have not yet been brought to light are of value to others working in this field. The Annual Meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION—to be held the week of May 5, 1941, in Pittsburgh, Pennsylvania—affords an opportunity to present papers on these subjects before a group which is most concerned with such developments.

In an effort to provide a balanced program of high quality papers, the Program Committee requests the cooperation of Association members. If you are at present engaged in work or know of work being done which should be reported at our meeting, will you communicate at once with GORDON C. HARROLD, Ph.D., Industrial Hygiene Laboratory, Chrysler Corporation, 7900 Joseph Campau Avenue, Detroit, Michigan, who is Secretary of the Association.

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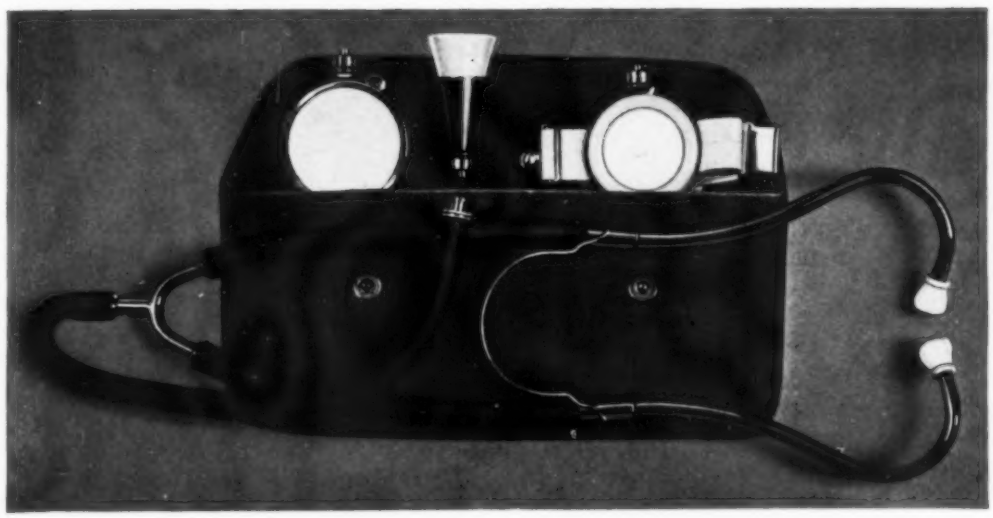

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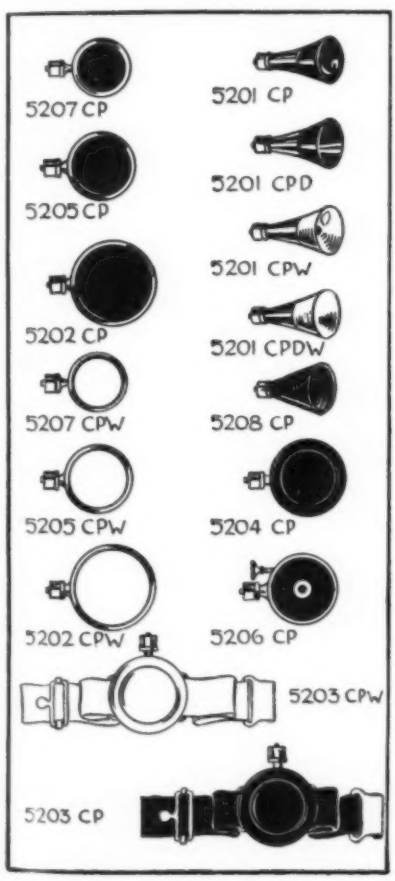

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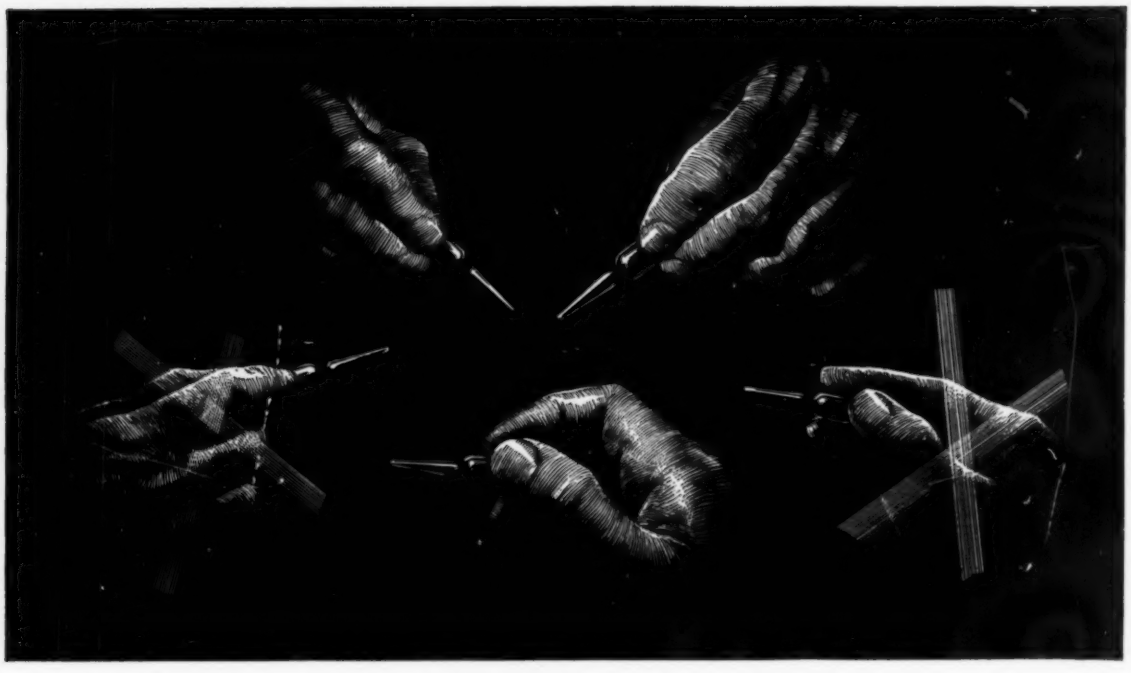

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